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AIDS TO HEARING.*

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The general public has acquired the belief that a hard-of-hearing individual need simply wear a hearing aid to bring his hearing up to normal and thus solve all his hearing problems. This is the idea conveyed by advertisements for hearing aids. Many physicians seem to share this idea. After all, if an individual has defective vision he usually can have it corrected by a properly fitted pair of glasses, and the two problems would seem to be similar. As a result there is a growing public resentment and intolerance towards the hard-of-hearing who do not wear hearing aids. Actually, there is no similarity between the two problems.

A hearing aid cannot be prescribed like a pair of glasses to bring sounds sharply into focus and correct hearing defects. The so-called tailor fitting of a hearing aid is largely a myth. A hearing aid simply amplifies sound like any public address system. It provides loudness or quantity, but not quality. The modern hearing aids do have adjustments that can suppress the amplification of low or high tones to a minor degree, but for the great majority of the hard-of-hearing, uniform amplification throughout the scale provides the best hearing ability. Not all hard-of-hearing persons can wear an electric hearing aid with benefit. This is especially true of traumatic deafness and presbycusis. If there be good hearing

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present for tones below 1,000 cycles but an abrupt nerve loss above that point, amplification of sound may improve the hearing intelligibility for voices but little, if at all.

Modern hearing aids are a boon to mankind and have reached a state of perfection and acceptance where they are providing useful hearing to millions of hard-of-hearing people. With a few exceptions the present vacuum tube aids are pretty well standardized, and the quality of performance of the different makes does not vary much. It would be wise for the purchaser of a hearing aid to ascertain the local facilities for servicing his hearing aid before making a purchase. Otologists should also obtain this information and should refer their patients only to those dealers who can provide repair or replacement service.

With the advent of transistors we can expect further improvements in the hearing aids of the near future. Elimination of the vacuum tubes will enable the aids to be made smaller; there will be no tube noises and it will be possible to increase the amplification of sound without the distortion that occurs with vacuum tubes. There will be a great economy of operation, as transistors require little current and the B batteries of the vacuum tube aids can be eliminated. The transistors are not perfected as yet, and occasionally break down without apparent reason. This is thought to be due to technical faults in their manufacture which are expected to be overcome in the near future.

The manufacturers and distributors of hearing aids are not helping the cause of the hard-of-hearing by their methods of advertising. *Do not forget that they are in a competitive business to sell hearing aids and make money.* The most effective advertising capitalizes on the psychologic weaknesses of the consumers. Much more stress is put on the size and invisibility of the hearing aids they sell than on the quality and fidelity of response of their instruments. They outdo one another in superlatives and in false implications. The Subcommittee on Audiometers and Hearings Aids in the A.M.A. has attempted to police this advertising with only indifferent success, and Better Business Bureaus are also helping in this respect. The final solution of misleading and improper advertising can only be reached when the hearing aid industry organizes to police itself. This it proposes doing, and we await results.

The major problem faced by those with defective hearing is the ability to converse with their fellow men. This paper is primarily concerned with the means of improving that ability. Electric hearing aids provide only loudness. The best hearing aid is one which provides this loudness with the least distortion. For those persons with a pure conductive type of deafness, loudness is all that is needed to enable them to hear distinctly. For those with mixed or nerve types of deafness mere loudness is not enough to provide distinct hearing. The greater the cochlear or nerve damage for tones within the speech range the less will be the power of discrimination for speech; moreover, because of the recruitment factor, too much loudness itself will reduce the power of discrimination. This is the reason why so many of the hard-of-hearing persons have difficulty in understanding conversation in the presence of much background noise.

I have a message to deliver to the hard-of-hearing and to those who deal with them as doctors or teachers, which might be called a philosophy for the deafened. In order to make my remarks authoritative and to explain this philosophy it will be necessary for me to relate some of my own experiences. I present my case history with no feeling of braggadocio, but merely in an attempt to prove what can be accomplished in the art of communication by people with serious hearing difficulties if they have the determination to overcome those difficulties and the willingness to make needed adjustments.

I have otosclerosis of the semi-malignant type with early cochlear involvement. There is no record of otosclerosis on either side of my family. My deafness first became apparent while I attended college, and increased rapidly. In 1917, after being discharged from the army because of my deafness, I entered medical school. Although I managed to complete the medical courses satisfactorily, my medical degree was temporarily withheld for the ostensible reason that my deafness might adversely affect my medical career and cause discredit to the institution. This was quite a shock to me, and marked the beginning of a period of depression and despondency which continued for some ten years. During this period I studied lip-reading, but I never became a proficient lip-reader. I also submitted to various operations and treatments which supposedly might improve my hearing. Surprisingly, I did seem to hear better following some of these procedures. Later I discovered that the apparently improved hearing was simply improved attention. Between times I had fallen into a common habit of the hard-of-hearing of not listening and I, therefore, seemed deafer than I actually was. I acquired a reputation for being a snob, because I often failed to reply to people who spoke to me. The medical practice which I had largely inherited from my father dwindled rapidly, and I became a subject of serious concern to my family. It was during this period that the carbon type of hearing aid appeared on the market, and I half-heartedly tried wearing one. I did not like it. Though I admittedly heard better, the distortion of sound and extraneous noises were very bothersome, and even with the hearing aid, I could understand only about one-half of what I heard; moreover, I felt conspicuous, and it was a severe blow to my pride to be advertising an infirmity which I had been trying to conceal for years.

A former patient of mine who proved to be a real friend did more to bring me to my senses than any other person. He told me that the reason he and other patients had left me and consulted other doctors was because of my attitude of indifference when they tried to tell me about their complaints; that I acted as though I were not listening to them, and as a result they could not determine whether or not I understood them. Consequently they were afraid that I would treat them improperly. This was quite true. I had thought that I was carrying on a successful bluff by pretending to hear what they said, whereas I actually was not even trying to listen to them half the time. I was rudely awakened to the fact that in trying to conceal my deafness I was fooling no one but myself; moreover, I was beginning to realize that my hearing was not going to improve with the help of any miracle treatments, but was actually growing gradually worse.

It was at this stage of my life that I finally accepted my deafness as an unalterable fact, and acquired the determination to overcome my handicap. I began to wear a hearing aid continuously, but without my former feeling of antagonism towards it. As I became used to the distortion and extraneous noises I found that my discrimination improved. I also noticed that if I wore the aid conspicuously people would speak to me a little more slowly and distinctly, and that fact alone improved my understanding for speech.

I began to use my eyes and my mind to help me listen and became increasingly adept as a speech reader, though I never did master the art of lipreading. If I can see the facial expression and catch some of the accented syllables, I can usually follow the trend of a conversation without much difficulty with the help of what sound I can hear through my hearing aid. I discovered that by wiring my hearing aid with a receiver for each ear I could understand much more than I could with a receiver in only one ear. This procedure, however, has its drawback. With both ears plugged by receivers it is very difficult to modulate one's own voice. It took nearly two years before I could control the modulation of my voice satisfactorily. This is probably the sole reason that so few individuals will wear a hearing aid with receivers in both ears, though I consider it a small price to pay for better hearing. When I became adjusted to my own disability my otologic practice increased rapidly, and many of my former patients returned to me. I soon discovered that my deafness, instead of being a handicap, was actually one of the biggest assets to my practice. Many deafened people consulted me because they believed I could understand their problems better since I also was hard-of-hearing.

I believe that one of the surest signs of the acceptance of and adjustment to a handicap or infirmity is the development of a sense of humor about that handicap. I, personally, take great delight from intentional misinterpretations of remarks made to me. A bizarre example of this occurred recently when I insisted that someone said that I was a nice, old bachelor when what he actually said was that I was a dirty, old bastard.

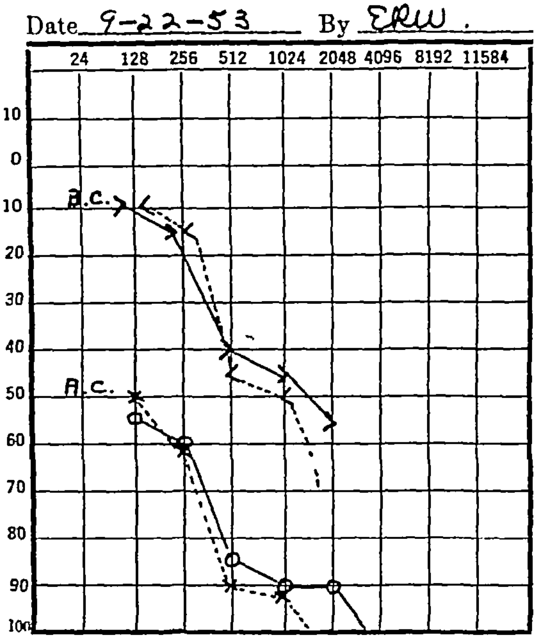

Fig. 1.

With the advent of vacuum tube hearing aids the quality of sound reproduction improved considerably, and much of the distortion which was produced by the carbon type of aids was eliminated. Although my hearing loss has increased somewhat in recent years I can understand more today with the present aids than I could 15 years ago. I believe that further improvement in quality will be attained with the transistor aids, aside from the reduction in their size and weight.

To complete my case history I am presenting a copy of my audiogram made just a few weeks ago by the Audiology Department of the University of Pittsburgh. (Fig. 1). Discrimination tests revealed that I have a 20 per cent discrimination score for my left ear and a 30 per cent score for my right ear for recorded voice with amplification of 94 decibels in a soundproof room. Using receivers on both ears my discrimination improved to 48 per cent. Greater amplification caused a sharp drop in discrimination. From these records it can be seen readily why I wear receivers in both ears.

My experience is not an unusual one, but is simply an example of thousands of similar cases, though my deafness is more severe than most. I have seen many a household disrupted because one hard-of-hearing individual would make no attempt to adjust to his deafness or accept any of the aids available for better hearing. This often has been due to lack of proper guidance or advice by his physician. In this modern era with the facilities available for helping the deafened there is little excuse for such an occurrence.

As a result of my own experience I have a few words of advice to give to the otologists. If you find that medical or surgical treatment is ineffective, do not continue to treat your deafened patients or give them hope that their hearing may improve. They will make no attempt to adjust to their handicap so long as they have any hope of improved hearing from treatment. Fully one-half of the maladjusted deafened people today have failed to make any attempt to help themselves, because physicians continue to hold out straws of hope for the restoration of hearing by means of miracles. It is an unpleasant fact that the great majority of our deafened patients cannot be helped by treatment, and we do them serious harm by continuing to treat them either in the hope of improving their hearing or with the false idea of preventing their hearing loss from growing worse. The longer that false hopes are extended to these people and necessary adjustments delayed the more difficult does it become to make these adjustments. On the other hand you will do these patients just as much harm by telling them that their hearing will get worse. The worst offense of all is to intimate that a patient may eventually become stone deaf.

Do not brush these patients off with the suggestion that they investigate hearing aids. They need advice and guidance, and it is the duty of the otologist to give it to them. Discuss their problems with them. Explain the use and limitations of hearing aids. Refer them to the local audiology department or rehabilitation center. Urge them to join the local league of the American Hearing Society, where they will meet other people more deafened than they are. There they will see for themselves how happy and cheerful the hard-of-hearing can be, and how well they converse after proper training. They can also get instruction in the use of a hearing aid and in speech and auditory training when indicated. The rapid development and growth of auditory centers in this country is an indication of the present recognition of the need for this type of work.

Finally I have a message for the severely deafened which I present as a philosophy for the hard-of-hearing.

Accept your deafness as an incontrovertible fact and acquire the determination to overcome the disability that it causes. Half way measures will not work. Complete acceptance is necessary before needed adjustments can be made.

Get yourself a hearing aid and wear it continuously as a part of your clothing. Do not try to conceal your hearing aid or the receiver in your ear. Discover how much it helps you to have people with whom you converse know that your hearing is defective. The average American is a courteous individual, and he will speak a little more slowly and distinctly if he sees that you are hard-of-hearing.

Be attentive and use your eyes and brain to help you listen. If your discriminative power is poor, you should study lip reading and get auditory training to help improve your hearing ability. You should wear receivers in both ears if by so doing your discrimination is improved, even though this entails the added problem of learning to control the modulation of your own voice.

Develop a sense of humor about your deafness. You will be surprised to learn that if you can laugh at yourself other people will laugh with you rather than at you.

Avoid situations where acute hearing is essential and you will be made to feel conspicuous. Take up a hobby to keep you busy and contented, and to replace activities where acute hearing is needed. Keep your mind actively occupied. A busy mind finds no time for brooding or self-pity.

Avoid the anti-social tendency of the hard-of-hearing. Get out and mingle with your friends and acquaintances and discover how ready they are to help you if you will only help yourself.

If you do these things you may be surprised to find that you are not really handicapped after all, and may thus attain peace of mind and tranquillity of spirit.

OCCLUSION OF THE ROUND WINDOW BY OTOSCLEROSIS.*†

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Occlusion of the round window by otosclerosis has become of clinical interest since the development of fenestration surgery. Occlusion of the round window along with stapes ankylosis has been found on histopathologic examination in a small number of cases, all of which were known to be profoundly deaf. Histopathologic proof that occlusion of the round window may occur without stapes and ankylosis has not been reported. The principal case to be described in this report demonstrates almost total occlusion of the round window without stapes ankylosis or involvement of the oval window region.

In cases of otosclerosis with the usual indications of stapes fixation and good cochlear function, the creation of a new fenestra over the ampulla of the horizontal semicircular canal has as a general rule allowed improvement in hearing up to approximately the 25 db. threshold level. An exception to this rule has been the occasional case in which, notwithstanding good bone conduction and other evidence of normal cochlear function, the hearing has failed to improve following the creation of a technically satisfactory window.

One such case history is briefly presented below. Preoperative and postoperative audiograms are illustrated in Fig. 1.

A. G., 398079. This white man, age 31, was first seen on Jan. 4, 1947. He complained of a gradual loss of hearing in both ears since the age of 16. A diagnosis of otosclerosis was made and a fenestration was done on the left ear on February 7, 1947. There were no operative or post operative complications. Although the fistula response has remained strongly active there has never been any appreciable improvement in hearing at any time after operation.

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† From the Division of Otolaryngology of the University of Chicago.

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Cases of this type have been observed by various fenestration surgeons and were discussed by members of the Otosclerosis Study Group in 1950.[1,2] Nilsson,[3] and Skoog[4] recently reported two such cases in a series of 53 fenestrations during which they performed accurate audiometric examinations at various stages of the operation.

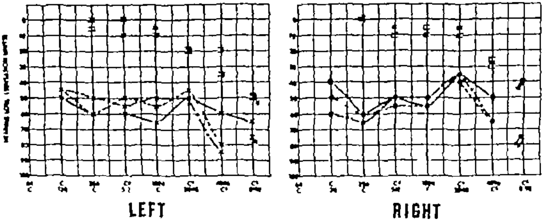

Fig 1 Bilateral otosclerosis Male, age 31 Fenestration, left ear, February 7, 1947. Pure tone air and bone conduction thresholds are shown for both ears preoperatively, six weeks postoperatively and six months postoperatively Note that the postoperative thresholds in the operated ear showed no significant improvement at any time

A probable explanation for failure to obtain an improvement in hearing in this type of case would be a total occlusion of the round window with a normally functioning oval window. Histopathologic proof has not yet been reported. Such an occlusion obviously could not be differentiated from stapes fixation by the routine tests now employed.

PHYSIOLOGICAL CONSIDERATION.

It is well established that the principal pathway through which sound enters the inner ear is by way of the oval window, and that the round window functions to permit mobilization of the incompressible cochlear fluids; however, it is evident that some sound must reach the round window through the air in the tympanic cavity. This would be in opposite phase to the normal stimulus. Lawerence,[5] however, has shown that this interference is negligible (about .8 db.). Under abnormal conditions the cochlea can be stimulated via the round window. Bekesy[6] has shown that after a radical mastoid operation, the sound may excite the cochlea through the round window route. Likewise several workers (Wever, Lawerence, and Smith;[7] Kobrak;[8] Kobrak, Lindsay and Perlman[9] have demonstrated cochlear stimulation by way of the round window in experimental animals.

Studies have been made on the effect on hearing of blocking the round window in animals. Hughson and Crowe[10] reported on improvement in hearing in cats when they blocked the round window with cotton. Wever and Lawerence[11] reported that all forms of blocking caused only minor variations in sensitivity; however, in a later series of experiments Wever[12] produced hearing losses of 25-30 db and interpreted the failure to obtain such losses in previous experiments as being due to incomplete occlusion. Wever stated that an air bubble of only .3 cubic mm. against the round window membrane would be effective in mobilizing the perilymph for normal cochlear function.

Measurements of bone conduction thresholds in the presence of an adequately blocked round window and a patent oval window have not been reported, as far as we know.

OTOSCLEROSIS OF THE ROUND WINDOW REGION.

The round window area is a common site for the occurrence of an otosclerotic lesion. Guild[13,14] states that "the round window region is next to the oval window region in number of otosclerotic areas. In 113 foci from 81 otosclerotic ears there were 25 lesions involving some part of the attachment of the round window membrane. Thus about one-fifth of all otosclerotic areas in his material were somewhere in this area. Guild further states that "only rarely do areas in this region develop into major lesions, i.e. into those that occlude the round window niche, or that otherwise might affect the acuity of hearing. A review of the chart of Guild's cases reveals that the otosclerotis process was confined solely to the round window area in only two of these ears.

Nylen[15] reporting on 121 otosclerotic ears found that 40 per cent of the foci involved the locality of the round window. In 8 per cent of the ears it was present in only the round window region.

Nager and Meyer[16] report that the round window is second to the oval window in frequency of otosclerotic lesions. In three of 53 ears the round window was the only area involved.

Nager and Fraser[17] published six interesting cases of extensive otosclerosis associated with otitis interna ossificans of the scala tympani. In three of these cases both windows were entirely obstructed. All cases had been profoundly deaf, the

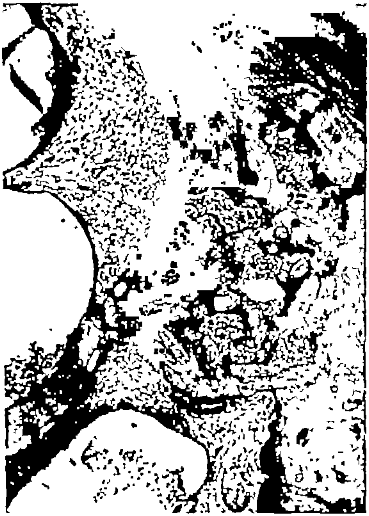

Fig. 2. Photomicrograph of a section through the attachment of the round window membrane and the spiral ligament, showing a minute focus of otosclerosis at the lateral attachment of the membrane. There is no encroachment on the round window. (RWM: Round window membrane.)

This case previously reported in *Archives of Otolaryngology*, Vol. 52, Dec. 1950.

explanation for which was not entirely clear since both degeneration of inner ear structures and occlusion of both windows came into consideration.

We have in our collection of temporal bones at the University of Chicago 37 ears which show otosclerotic lesions. Seventeen of these ears have foci in the round window area. In four ears from three cases the process is exclusively confined to this region. There is a small circumscribed active focus of otosclerosis situated at the lateral attachment of the round window membrane in three of these ears (Fig. 2) obtained from two cases. These foci undoubtedly produced no loss of hearing.

The one ear which was obtained from the third case showed a single focus of otosclerosis occluding nearly all of the round window and will be presented in some detail.

Mrs. B. B., 262696, a 35-year-old white professional singer was under the care of Dr. Ruth E. Taylor of Chicago from 1944 to 1951. When first seen she complained of recurring attacks of pain down the right arm and left chest present since childhood, but worse since a pregnancy in 1941. Nodules were noted about the tendon attachments on the hands and feet. Later she developed substernal pain on exertion, and clinical evidence of arteriosclerotic heart disease. Family history revealed that her mother had died from cardiac disease and that her three brothers suffered from a similar condition.

Blood lipid studies were done on Mrs. B. at the University of Chicago in 1949. The blood cholesterol was 426 mgms. per cent and the total blood lipids were 1155 mgms. per cent. A diagnosis of familial xanthomatosis was made. The same diagnosis has been confirmed in the cases of the three brothers, two of whom have since died of coronary occlusion, while the third has presented symptoms of coronary insufficiency.

During 1947 the patient was seen several times because of a vocal nodule. There were no complaints or abnormal findings referable to the ears. She was aware of the nature of her disease and alert to any new symptoms. She was under close observation by her physician up to the time of her death, but at no time mentioned any auditory symptoms.

On December 24, 1951, she died suddenly when walking in a heavy snow in a blizzard. An autopsy was performed on December 25, 1951, by Dr. Eleanor Humphreys. The findings may be summarized as follows: Familial xanthomatosis tuberosa with xanthofibrous nodules and cholesterol granulomas on the extremities. Severe arteriosclerosis of the aorta and coronary arteries. Multiple scars of small infarcts and extensive patchy fibrosis in the myocardium. The cause of death was acute coronary insufficiency.

The right temporal bone was removed and presented to the Division of Otolaryngology for histopathologic study. The bone was fixed and decalcified in the usual manner. Serial horizontal sections were made 20 microns in thickness.

Histopathology. See Figs. 3 to 6.

Middle Ear. The tympanic membrane, ossicles and mucous membrane were normal. Pneumatization was normal.

Inner Ear. The membranous structures were in a fair state of preservation, but showed moderate degree of postmortem degeneration.

Cochlea. The cochlear duct was normal except for moderate post mortem degeneration of the organ of Corti. The spiral ganglion and its axons and dendrites were normal. The cochlear nerve was normal. The vestibule, the utricle, saccule and semicircular canals were within normal limits.

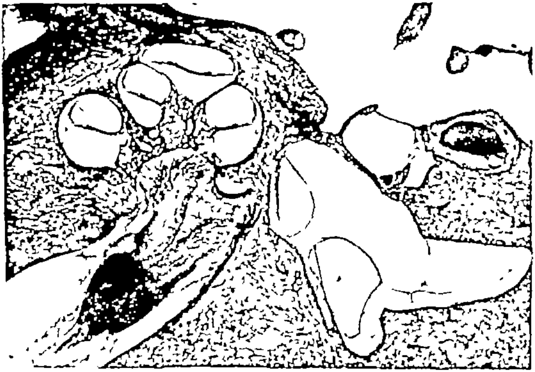

Fig. 3 Horizontal section through the cochlea and vestibule at the level of the oval window There was no otosclerotic lesion at the site of predilection, or any part of the otic capsule other than that shown in Fig. 4

Otic Capsule. The periosteal, enchondral and endosteal bone was normal except for an area of typical otosclerosis in the round window area. There was no evidence of otosclerosis in the site of predilection at the oval window or elsewhere in the otic capsule.

The lesion occupied nearly the full thickness of the capsule below the spiral ligament. The round window was completely obstructed in its superior part. A small medial portion remained free, and connected inferiorly by a narrow channel to the middle ear cavity. The mucous membrane adjacent to the focus was thickened and contained many dilated blood vessels, some of which connected directly with vessels in the otosclerotic focus. This hyperemic mucous membrane further obstructed the open portion of the niche. There was a fracture extending from the posterior edge of the focus to the posterior bony semicircular canal beneath the crista ampullaris, a region in which a small fracture is frequently seen in temporal bones.

A graphic reconstruction of the round window region was made in order to estimate the area occluded by the focus. The total area of the round window was found to be 1.9 sq. mm. The area of the membrane which was free from otosclerotic bone measured 0.38 sq. mm. Mucous membrane containing many congested vessels, further reduced the remaining air space in the niche. The measurements showed that at least 80 per cent of the round window was obstructed by the otosclerotic bone.

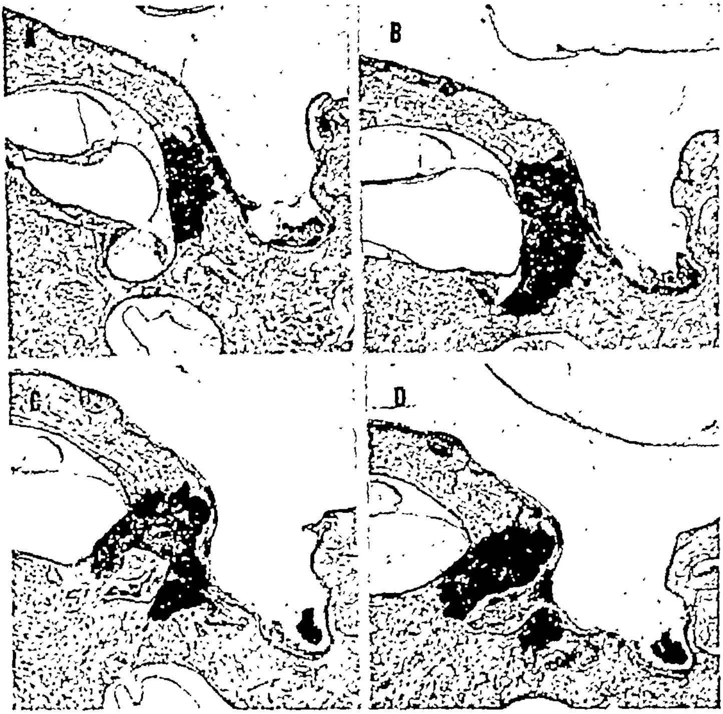

Fig. 4-A. Horizontal section through the basal coil, through the upper aspect of the otosclerotic lesion which lies on the lower promontory adjacent to the spiral ligament at this level.

Fig. 4-B. Section lower than 4-A. The focus occupied almost the full thickness of the capsule. The round window membrane is seen adherent to the otosclerotic focus, except at its medial attachment.

Fig. 4-C. Section below 4-B showing complete occlusion of the round window area at this level. A small portion of the niche remains patent.

Fig. 4-D. Still lower section showing the small remaining communication of the niche within the middle ear.

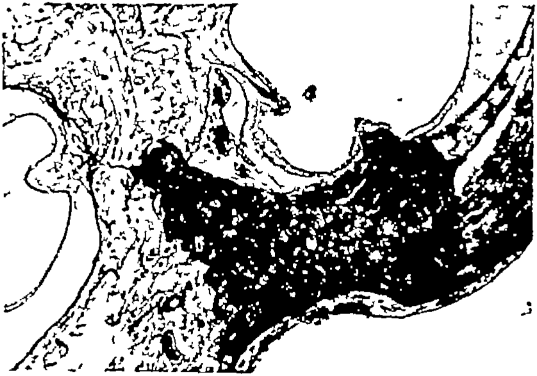

Fig. 5 Higher magnification (x 32) of the lesion. The round window membrane is adherent to the focus anteriorly, but at this level it is free posteriorly. The mucous membrane on both sides of the focus is considerably thickened and contains many dilated and congested blood vessels.

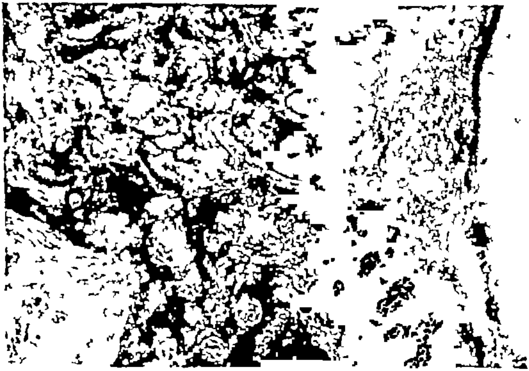

Fig. 6 High power (x 135) showing the typical histopathological appearance of the otosclerotic lesion. The superior half appears sclerotic and inactive while the inferior portion shows many dilated vessels indicating activity.

DISCUSSION.

The case just described provides histologic proof that occlusion of the round window by otosclerosis without involvement of the oval window may occur. The extent and the degree of activity apparent in the focus in this case leaves little doubt that it would have progressed to complete occlusion, had the patient lived. This patient can safely be assumed to have had good hearing until the time of her death. The absence of hearing loss corresponds with the observations made by Wever, who stated that if a small area of the round window membrane remained free the hearing would not be affected. In this case 0.38 sq. mm., equal to 20 per cent of the total area, was free from the otosclerotic focus.

Wever found that occlusion of the round window could cause air conduction loss comparable to that produced by stapes ankylosis. It seems probable that bone conduction thresholds would not be raised correspondingly, although experimental evidence on this point is not available.

If occlusion of the round window is capable of producing air and bone conduction thresholds similar to those found as a result of stapes fixation some method for differentiating the two conditions is needed. Theoretically the principle involved in the Gelle test offers a possible approach, and with modifications to provide for greater precision of measurements might prove to be of practical value. The mobility of the stapes, as determined by testing with the probe during the fenestration operation, may not be a sufficiently accurate indicator of the efficiency of the mechanism as a sound conductor.

SUMMARY.

A case of otosclerosis has been presented in which the focus has been limited to the round window region and has produced occlusion of 80 per cent of the round window membrane.

The absence of hearing loss in this case corresponded to experimental observations made by Wever on the degree of occlusion of the round window necessary to cause hearing loss.

The histologic evidence of activity in this focus suggests that complete occlusion would have eventually developed.

This case confirms the possibility of occlusion of the round window by otosclerosis in the presence of a patent oval window.

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BONE GRAFT FOR ATROPHIC RHINITIS.

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The purpose of this work has been to devise a surgical technique which would tend to restore "normal" nasal function in patients with atrophic rhinitis.

The etiology of atrophic rhinitis has remained obscure. Medical treatment with vitamins, antibiotics, hormone sprays and other drugs has not only failed to improve the condition but even to halt its progress. The surgical management of this condition, however, has been more promising.

Nearly all surgical procedures devised have been directed towards narrowing the functioning airway. It has not yet been found possible to reverse permanently, even in part, the insidiously advancing chronic inflammatory reaction that involves the nasal mucous membranes and the cartilage and bone of the nose, both internally and externally.

Gersuny[1] injected paraffin submucosally, but his results were poor, due to extrusion and spread of the material. Lautenschlager[2] approached the lateral nasal wall via the Caldwell-Luc incision and mobilized this wall medially. The success of the operation depended on synechia between the septum and the turbinates to narrow the air space. Halle[3] modified Lautenschlager's technique, as did Hinsberg.[4] All of these operations, however, interfered with nasal physiology.

Aubonne[5] suggested the use of cartilage and bone implanted subperichondrially in the septum in an effort to narrow the airway. Eisenstodt[6] also suggested the use of cartilage subperichondrially. Beck, quoted by Ersner and Alexander,[7] sug-

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gested the use of bone splinters from the patient's rib, which were placed under the perichondrium of the nasal septum. Zorzoli[8] recently used bovine cartilage in the same way.

Osteotomy of the nasal bone followed by narrowing of the nasal pyramid has been suggested by Ersner and Alexander[7] and Kopp.[9] Narrowing of the nostril and movement of the lateral mucosal wall, together with the inferior turbinate toward the midline, has been suggested by Cottle[10] as another facet of the design for improvement.

Proud,[11] Guttierez,[12] and Causse[13] reported the use of acrylic resins and other plastics in the septal space as a satisfactory method for narrowing the nasal chambers. Kasnetz[14] and Kemler[15] implanted ivory in the same way.

The use of acrylic resins as septal implants has the advantage of making a relatively simple procedure out of the operation. With the Proud technique, it has produced improvement in a number of patients; however, one fears that the pressure of an inert substance will adversely affect the nutrition, and then cause further damage to the nasal mucosa. Inert substances, such as acrylic resin, are also likely to be extruded. Once the mucous membranes have healed following extrusion, dense scarring impairs any future chance to insert another substance. The danger of extrusion also exists with substances such as glass, ivory or metal.

Cartilage and bone used as implants have failed in the past because isogenous or autogenous bone has either been absorbed if tolerated or has been extruded when placed under the perichondrium adjacent to septal cartilage. Resorption rather than extrusion of the cancellous bone took place even when most of the septum has been resected. Cortical bone also behaved in the same way. Cottle[10] has previously used cancellous bone into the septal space and palatal bed area.

In the past, failure of bone grafts to "take" has led to the method's being discarded. A necessary prerequisite of a successful "take" is an adequately prepared bone bed. Atrophic rhinitis involves not only the mucosa and submucosa, but the underlying bone as well; therefore, an adequate blood supply for the bone graft was difficult to obtain. It was felt, however, that if a bed could be prepared in the palatine bone, the blood supply would be better and a "take" more likely to be secured. If a successful "take" of a cancellous bone graft to the palatine bed could be accomplished, it was reasoned that this graft could assist in the nourishment of the septal mucosa and a more normally functioning mucous membrane. Since a good deal of the septal mucosa is nourished by mucoperiosteum, I have attempted to evaluate the effect of a cancellous bone graft placed in the septum on a bed formed by the palate bones in narrowing the airway and of producing an improved blood supply to the mucoperiosteum of the septum. To exclude other factors which might influence the result, moving the lateral nasal wall towards the midline, narrowing the nasal pyramid or the nares was not done.

Surgical Technique: The operation is divided into two parts: the first consists of securing a cancellous bone graft. The largest piece of cancellous bone available is from the iliac crest. Accordingly a large segment measuring at least three by six centimeters including the entire cortical thickness is removed. All cortical bone is removed from the specimen secured and only the cancellous portion is used. I have facilitated the operative procedure by having another surgeon perform this part of the operation at the same time that the nasal part of the operation is being done.

The second procedure is the operation on the nasal septum. An incision is made over the caudal end of the septum and is carried down onto the floor of the nose and laterally into the ala in order to facilitate admission of the highest and longest graft possible. The caudal part of the septal cartilage is left, but an extensive septal resection is performed leaving only a bar of cartilage under the lateral cartilage. Then using a four millimeter chisel, a deep trough is cut into the hard palate from the anterior nasal spine backwards. The mucous membranes are then separated for a distance along the floor immediately adjacent to the trough, so that no tension will exert on the mucous membrane flaps when the graft is inserted. The graft is then placed in the trough.

The graft is made wide enough that the septal mucous membrane flaps on each side touch the inferior and middle turbinates. The incision is closed with silk. No packing is used.

Following the operation the patient is placed on antibiotics and given crutches to get about. Usually the patient can get about fairly well without the aid of crutches in a week.

The following cases are presented to indicate the results obtained by this procedure:

Case 1: S R., 51, white, female Diagnosis Primary Atrophic Rhinitis with Ozena. Acrylic implant was done by the Proud technique in 1950. There was some symptomatic improvement. Anosmia with minimal crusting remained, and there was no objective change in the nasal mucosa. The

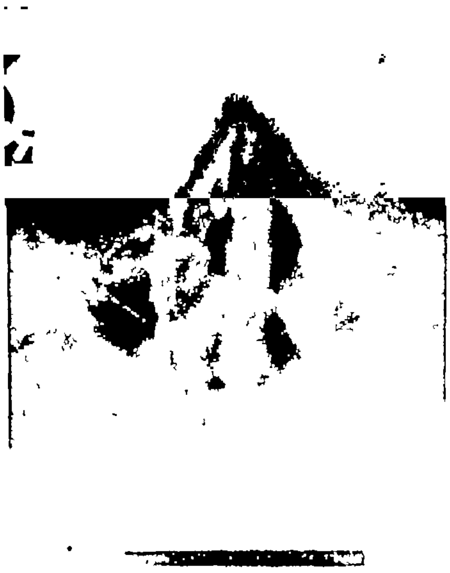

Fig 1 Two years after surgery Note the delicate bony trabecular and height of the graft.

patient attributed a weighty feeling in her head to the implant. The implant was removed six months after its insertion and a large single piece of cancellous bone from the hip was placed into the septum in contact with a freshly prepared trough in the bony palate. The septum was widened nearly to the point of objective nasal obstruction, and the patient complained of nasal obstruction for the first three months post operatively.

At present, the nasal mucosa has a normal pink appearance. This change has affected the mucosa over the turbinates as well as that of the septum. The middle and inferior turbinates appeared larger and pinker. Several otolaryngologists who have seen this case without knowing about the procedure have remarked that she needs a "submucous resection." The patient remarks that "I can breathe better than at any time in my life and now I can smell my food." Figs. 1 and 2 were taken two years after surgery. The six-months' X-ray showed the graft to be very slightly larger than the present, indicating that some resorption had taken place later.

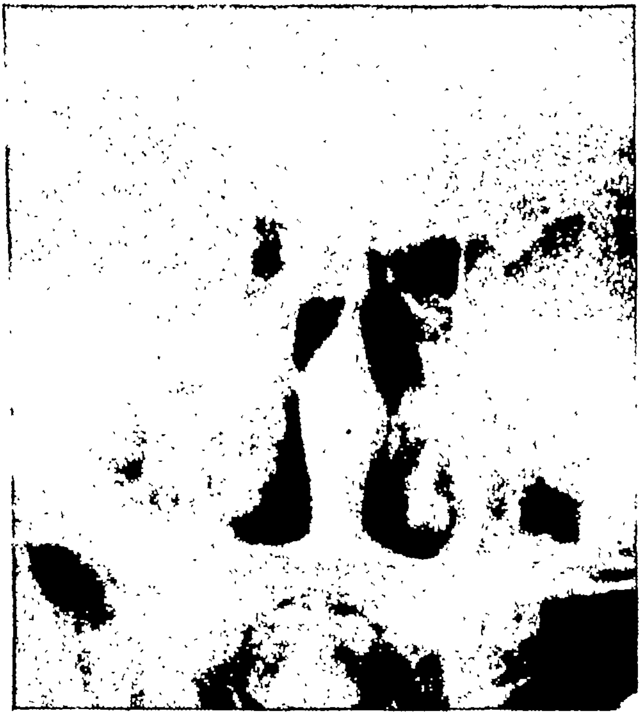

Fig. 2. Two years postoperative. The fusion of bone graft with palate is distinctly visible.

Case 2: K. M., 17, white, female. Diagnosis: Atrophic Rhinitis and Bilateral Chronic Maxillary Sinusitis. Preliminary bilateral Caldwell-Luc and transantral ethmoidectomy was done when conservative treatment

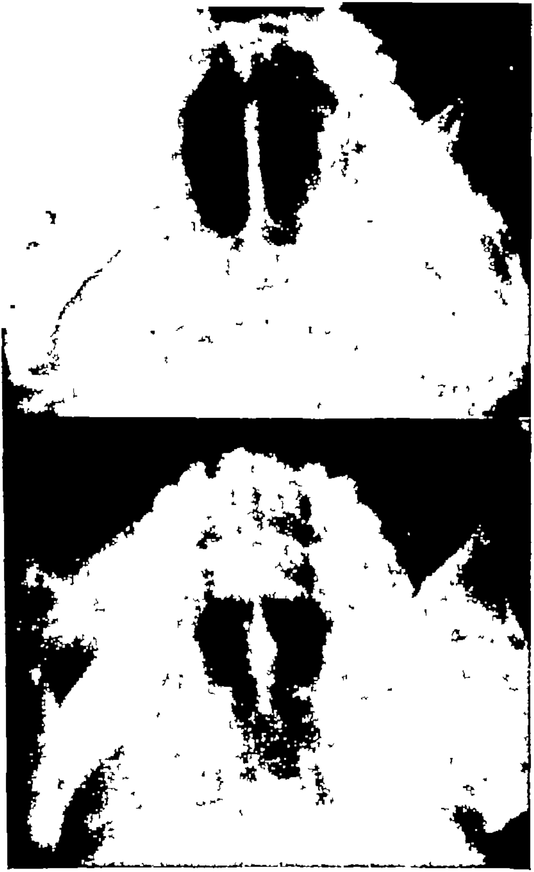

Fig 3 Submentovertical position The upper view is a preoperative picture. The lower view, taken two years after surgery, shows a large healthy graft extending posteriorly to the level of the front face of the sphenoid.

failed to improve the condition. One month later a piece of iliac crest was removed. At the time of surgery, it was noted that the cancellous bone was thin and not well developed, and portions of it appeared to be cartilaginous. A single large piece could not be obtained, and even with the insertion of several small pieces of cancellous bone, nasal obstruction could not be obtained. At two months the airway looked ideal, but at three months, it appeared more open and the turbinates remained small and atrophic. On comparison with the previous case this indicated that insufficient bone was placed to narrow the airway sufficiently and with resorption the space became too open. At one year, symptomatically, there was only fair improvement, but objectively there was no change in the appearance of the nasal mucosa.

Case 3: G. C., 58, white, female. Diagnosis: Primary Atrophic Rhinitis and Cylindrical Bronchiectasis. At the time of the removal of the piece from the iliac crest, the cancellous bone was noted to be thin, scant and osteoporotic. Sufficient bone to occlude the airway could not be obtained, although a satisfactory bone bed could be made in the hard palate. Resorption proceeded as rapidly in this instance as in case 2. Symptomatically, she had fair improvement, but objectively there was no significant change.

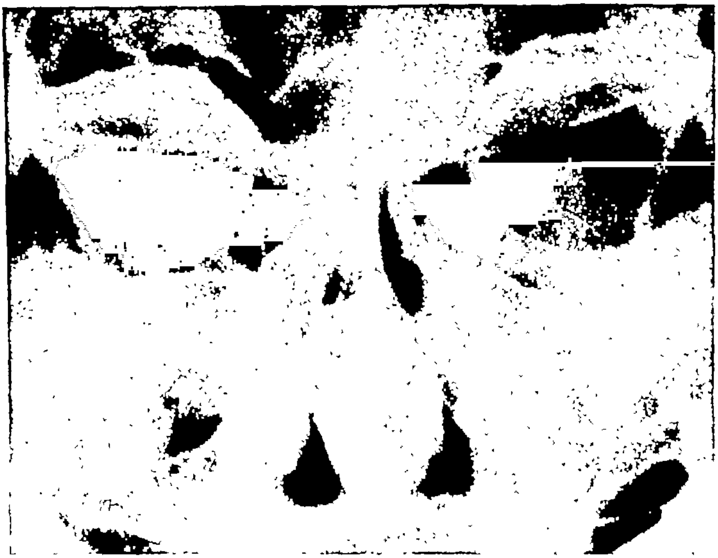

Fig. 4. The graft is a good deal wider at the middle turbinate meatus area.

Case 4: B. R., 29, white, male. Diagnosis: Primary Atrophic Rhinitis. Cyst of right maxillary sinus. The cancellous bone was excellent, and a piece wide enough to obstruct the nasal chambers was placed in the septum. It took fully four months before the nasal chamber opened sufficiently to produce subjectively normal nasal respiration.

It is now two years post-operative (see Figs. 3 and 4). The patient states that he can breathe very well and has no sense of obstruction. Crusting is absent. The nasal mucosa is pink over the septum and the middle and inferior turbinates appear larger and healthier. This appears to be an excellent result.

DISCUSSION.

Two years have passed since the first of these four cases was operated upon. It has appeared that in selected instances, bone grafts to the septum have been satisfactory in relieving the symptoms and signs of atrophic rhinitis. In the patients with good results, not only is there symptomatic improvement, but also the pale thin mucosa is changed to a more normal pink turgescent appearing membrane over the septum and turbinates. It is interesting that the narrowing of the nasal chamber by widening the septum with living bone causes the atrophic mucous membrane of the lateral nasal wall to approach a more normal color.

A properly prepared bony bed in the hard palate is a most essential requisite for success. The characteristics of the cancellous bone and the amount available also seem to be an important factor in securing a good result. Results with the use of iliac bone containing cartilage in the younger individual with *chronic purulent sinusitis, or in the older person* with osteoporotic bone indicate that at the present time we cannot expect a good result from this procedure alone.

Healthy and adequate iliac bone should be placed into the septum until nasal obstruction is obtained. Some resorption takes place during the first three- to four-month period before physiologically adequate airway is established. The insertion of cancellous bone into the septo-palatal area appears to be a reasonable and sound method for the relief of atrophic rhinitis. Further time will be required to tell whether the improvement gained will be permanent.

One case operated upon by the above technique and of less than two years' duration was not included in this report. To date, the result has been excellent.

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THE "FALSE-BING" PHENOMENON—SOME REMARKS ON THE THEORY OF BONE CONDUCTION.

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There can be no doubt that in all cases of pure conduction deafness, the bone conduction audiometric curve for the low frequencies is normal or very slightly below normal curve. All audiometricians agree with Professor Carhart's schematic curve[1] which shows a very smooth slope between 256 and 2048 cycles, where the maximum mean loss is about 15 db (see Fig. 1-a).

To begin with, we think it necessary to point out that Carhart's curve contradicts the presently accepted theory of bone conduction, which proceeds from the experimental findings of Barany and O. Bekesy. It is quite obvious that if, for the low frequencies, bone conduction is due primarily to the inertia of the ossicular chain, the fixation of the stapes should be translated on the audiogram by considerable loss up to about 1000 cycles. On the other hand, if, for the high frequencies, bone conduction is due primarily to the compression of the bony labyrinth, above 1000 cycles the effect of the compression of the semicircular canals should be translated by supranormal bone conduction. "We can suppose," writes I. Hirsh[2] "that if the stapes is fixed, as it is in otosclerosis, bone conduction will be better than normal, but primarily for the high frequencies when compression bone conduction plays the major role." In fact clinical otosclerosis is characterized by a bone conduction curve which according to theory is the exact contrary of what could be expected.

Let us consider a case of unilateral conduction deafness. We suppose for instance that the right ear is defective, with a bone-conduction curve normal, or perhaps slightly below

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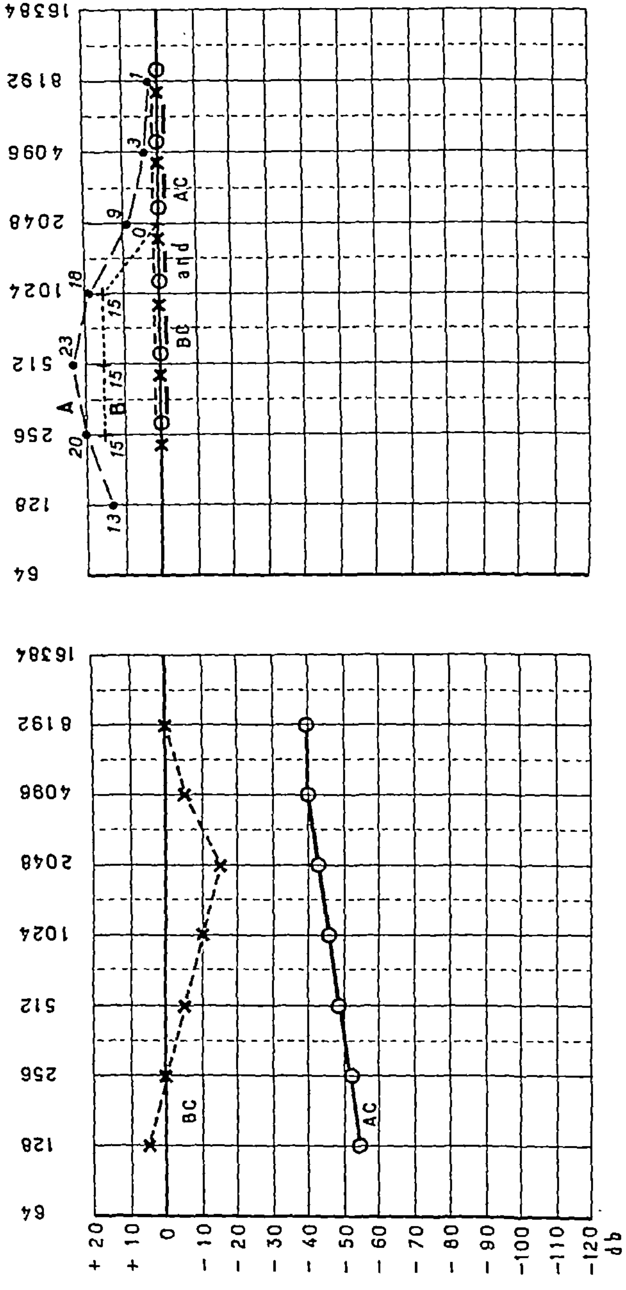

(b)

(a)

Fig. 1. a. . . Schematic bone conduction audiometric curve in case of stapes fixation (according to Carhart). b. Shift of bone conduction thresholds by occlusion of the Ear canal. A—wet cotton batting inserted (data from Sullivan, Gottlieb and Hodges). B-finger inserted.

normal on the low frequencies, and a left ear absolutely normal for the same frequencies. The Weber test shows that the bone conduction sounds are heard and localized in the right ear. Why should it be so, since at most both ears are normal as far as bone conduction is concerned? As far as we know, this has never been answered; in fact, one generally believes that if, in unilateral pure conduction deafness, the patient lateralizes bone conduction in the bad ear, it is because on that side, bone conduction is better than in the good ear: that is, better than normal.

It is easy to prove that this is not the case. To demonstrate, we must refer to the work of Sullivan, Gottlieb and Hodges on the occlusion shift for normal ears.[2] When the normal ear is occluded by means of wet cotton batting inserted in the canal, the bone conduction thresholds are shifted significantly above normal (see Fig. 1-b). We personally have no experience of this method of occlusion, but we have a wide one of the simpler method which consists in closing the ear with the finger, and, in such a case, the bone conduction shift is slightly below Sullivan's figures; it is about 15 db on the three frequencies, 256, 512 and 1024 cycles, the "0" index being between 30 and 59 db.

In a case of unilateral pure conduction deafness, if one occludes the good ear, it is evident that on that side bone conduction is significantly better than in the bad ear. The Weber test should then be lateralized in the good ear but, in fact, it is not. In all cases of unilateral pure conduction deafness of more than 30 db—and in 85 per cent of all cases—when one proceeds to close the good ear, the Weber remains lateralized to the bad ear. Moreover, and we consider this as very important, the occlusion of the good ear produces a significant shift of the bone conduction threshold of the bad ear, objectively as well as subjectively (see Fig. 2).

This, by analogy with the "False-Rinne negative" we have called the "False-Bing" phenomenon. It happens in all uni-

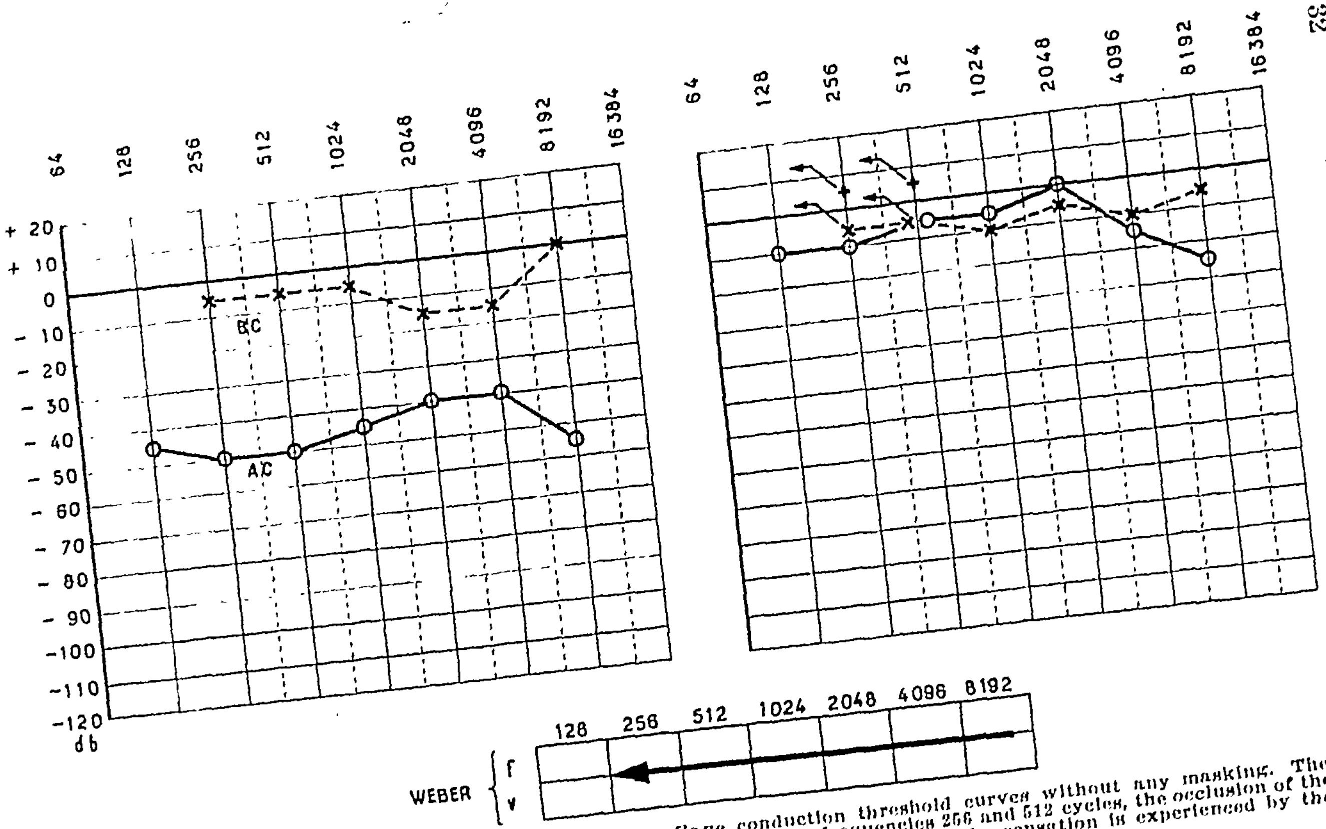

Fig. 2. A case of unilateral pure conductive deafness. Bone conduction threshold curves without any masking. The Weber is lateralized on the right side. As shown by the arrows on the frequencies 256 and 512 cycles, the occlusion of the left normal ear has produced a shift of 10 db in the bone conduction threshold, and the sensation is experienced by the patient in the defective right ear.

lateral pure conduction cases where the deficiency of the bad ear is above 30 db. One cannot always detect it for all the low frequencies; the probabilities of occurrence are:

256 c.	80 per cent.
512 c.	83 per cent.
1024 c.	43 per cent.
2048 c.	3 per cent.

Above 2048 c. the "False-Bing" phenomenon never occurs.

It should also be noted that this phenomenon occurs only in pathological cases. It does not happen when one tries to simulate a unilateral pure conduction deafness by occluding one ear with a finger. In that case the occlusion of the contralateral ear with a finger will easily and progressively reset the Weber in the center of the head.

Incidentally this experience shows that one should not consider that the Bing test (occlusion) and the Weber test are two different aspects of the same phenomenon. In Germany the terms applied to both tests make the confusion absolutely complete; in that country, the Bing test is called "Physiological Weber, because it seems that it was discovered at first by Weber and not by Bing, and the Weber test is called "Pathological Weber." The Bing test produces a significant shift of bone conduction threshold on the low frequencies, while the Weber test, in pathological cases, does not. The Bing test is, therefore, perfectly explained by O. Bekesy's hypothesis on the inertia of the ossicular chain, the effect of which is enhanced by the inertia of the head of the mandible vibrating in a closed canal. The Weber test is not explained by such a hypothesis.

What we have said above concerning the "False-Bing" phenomenon, shows that the Weber test remains unexplained: in unilateral pure conduction deafness, the Bone conducted low frequencies are heard in the bad ear, although we know for certain that on that ear bone conduction cannot be better than in the opposite good ear.

From the practical point of view, these findings have important bearings. It is utterly impossible in cases of unilateral pure conductive deafness to mask the bad ear in order to measure the bone conduction thresholds of the good ear, whatever may be the amount of white or saw tooth noise produced. Besides we may add that the present methods of calculation of the amount of noise necessary to mask an ear, do not take into account the fact that the bone conduction curve of the bad ear may be lower than the bone conduction curve of the good ear.

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HISTAMINIC CEPHALALGIA.*
(SUICIDE HEADACHE).

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DEFINITION AND DESCRIPTION.

Histaminic cephalalgia is a two-phase unilateral, agonizing headache, occurring mostly at night or when the patient is relaxed, and results from rapid dilation of the external carotid artery. In typical cases, the pain areas follow the anatomical distribution of the artery. The attacks begin suddenly and are short, but frequently recur three or four times a night.

This headache is distinguished from migraine, which is a three-phase, unilateral headache beginning with vasoconstriction (when warning signs of impending attacks are present) followed by vasodilation and prolonged perivascular edema, which may persist for many hours or days.

I have called the syndrome "suicide headache" because so many of my patients have said that if they could not get relief, they would "end it all."

Patients will rave, scream, hit their heads against the wall; some will strike their heads with various objects in a futile attempt to deaden the pain. Since there is unilateral lacrimation during the severe pain, I have added another definition, "The headache of the raving people who weep with one eye only." This is descriptive of the extremely severe type of histaminic cephalalgia.

The following case history is illustrative:

H. H., age 46 years, a motor car executive, had terrific unilateral night headaches for six years. Each attack was accompanied by unilateral lacrimation, nasal obstruction, and other symptoms. The attacks would

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last thirty minutes to two hours. He seldom escaped an evening or a night without one attack—sometimes three or four. He had noticed that his attacks came only when he was relaxed after work in the evening or they would awaken him out of a sound sleep. While sitting with his family in the evening watching television or listening to the radio, his children would become frightened when they saw his right eye begin to lacrimate. The children would dash out of the room or go to the neighbors until the screaming was over. He always appeared to be in a violent rage, but never injured anyone except himself. He seldom slept more than four hours; by morning, relief would come when he had to return to work, utterly exhausted.

He developed two methods of counter-attacks: 1. When the pain would start he would jump out of bed screaming, dash out of his home into a nearby park, dressed only in night clothing, even in severe winter. His wife would follow with slippers and robe. He would run and scream until the attack was over. The screaming and running seemed to lessen the severity of the attack. 2. He would hit himself on the head with a hammer in the vain hope of deadening the pain.

In the six years he had received 13 different diagnoses from 26 doctors in six states; several of the physicians were heads of departments in large teaching institutions. The most common diagnosis was a neurosis, always with the advice to have psychiatric treatment. Several times the condition was diagnosed as "sinusitus" and he had one unnecessary sinus operation. He was sent to a hosiptal one night where an exploratory intracranial operation was advised. Fortunately the operation was not performed, as the pain stopped suddenly. Other diagnoses were migraine, glaucoma, syphilis, pinched nerve, and bad teeth. Each physician consulted felt sure he understood the trouble, and various treatments, which never did any good were prescribed, always starting with morphine for the acute episode.

With the housing shortage following the war, he had great difficulty finding a place to live because of his nightly screaming episodes. He always tried to rent an apartment near a park or an athletic field, where he could run and scream without disturbing too many neighbors or attracting the attention of the police.

More than two months were needed to get this patient stabilized on the correct dosage of histamine, and other treatment, before he was relieved. During this period he was given a letter authorizing any physician, intern, resident or nurse to administer DHE_{45} intravenously. Frequently he would be relieved from the acute attack before the needle was out of his vein. His wife would drive him to the emergency room of the hospital; he could always drive home. He carried his own DHE.

ETIOLOGY.

The etiology of histaminic cephalalgia is unknown; however, I have developed a working hypothesis based only on clinical observations over more than ten years and with more than 100 patients. This hypothesis includes primary and secondary causes, and I will discuss the secondary first. The disease, I believe, is due to a secondary involvement of the autonomic nervous system, the impulses from which are mediated through the great control center, the hypothalamus. The hypothalamus is the control tower that regulates the rhythmical contractions and dilations of all blood vessels, as well as the body rhythm.

The severe pain of histaminic cephalalgia is produced by an unusual dilation of the external carotid, which stretches all the surrounding sensory nerves and tissues. In the typical case the pain locations follow almost the exact areas supplied by the artery. The pain lasts as long as the artery remains expanded. The instant the artery regains its normal size the pain stops, which can occur in a fraction of a second, but more often there is a "fading out" period of a few minutes to half an hour.

There is little doubt that there is a release of histamine in the body just before the pain starts. Also, there is little doubt that there is an increased body sensitivity to histamine, as well as sensitivity to other allergens. I have been unable to prove to my own satisfaction that histamine sensitivity can be demonstrated by cutaneous testing to the extent that would be of any real diagnostic value. Others have contrary opinions.

It is assumed that there is a sudden release of histamine in the body followed immediately by dilation of the external carotid artery resulting in excruciating, unilateral head pain. It may be assumed, also, that a disturbed vascular rhythm is transmitted from the autonomic nervous system to the hypothalamus, the great regulator of vascular rhythms. Notwithstanding both of these physiologic assumptions, we still do not know the primary cause of the autonomic dysfunction —this disturbed rhythm.

Supported only by clinical observations, I believe that an imbalance or deficiency in electrolytic metabolism develops a nutritional or hormonal disturbance. If one would successfully manage this devastating disease he must be able to visualize the probable primary and secondary causes and combat them energetically for years.

SYMPTOMS AND DIAGNOSIS.

Certainly the diagnosis cannot be complete without an intensive, physiologic and analytic investigation of these phases. Women have said that their headaches, their allergies, even asthmatic bouts disappeared completely during pregnancy—especially during the seventh and eighth months when the estrogenic blood levels are the highest. Physicians have not given these voluntary statements the meticulous attention warranted by the physiologic facts observed. I am now making a vigorous and methodical investigation of all suspected hormonal disturbances—both estrogenic and androgenic.

Since the diagnosis is made almost entirely by symptoms, I will repeat the story told by a patient with typical histaminic cephalalgia:

> A young woman executive said on her first visit, "You are the last physician I am going to consult. I work hard all day and go home at night so exhausted I fall in bed, often without eating dinner. I am awakened out of sound sleep from one to five times a night with a pain so severe I have to jump out of bed. It is as though someone was plunging a knife into my right eye, my right temple, and the back of my neck. My head and eyes feel as though they would burst, the pain is so intense. The attacks last from 15 to 45 minutes; then all is well until the next bout begins in one or two hours. Of course, in the morning I am more exhausted than when I went to bed. I can't take it any longer!"

This intelligent young woman gave a classical history of a typical histaminic cephalalgia. When questioned further she described the characteristic unilateral nasal obstruction, lacrimation, flushing and, of course, redness and swelling of the right eye, all of which disappeared abruptly when the attack was over—only to recur when the next painful episode began.

Not all cases of histaminic cephalalgia are typical; not all patients give an intelligent history. Many will say, "I have terrible headaches," and leave it to the physician to find out as best he can the correct diagnosis. In a busy office or clinic the patient does not always get a chance to tell the whole story. Without the story a correct diagnosis is seldom made. It is easy to say, "You have a tension headache" or "a nervous headache" or "change your way of life," which the patients seldom can or will do. It is easy for an otolaryngologist to see a deviated septum, or perhaps a cloudy sinus, and otolaryngologists are often abetted in their misconception by roentgenologists who will find a slightly cloudy sinus or, as they frequently report, "thickened mucous membrane in a sinus suggesting an old chronic infection." This has, in the past, been sufficient evidence to start a march on a series of unnecessary and often mutilating surgical procedures on sinuses. Horton[1] says that over 40 per cent of his patients have had unnecessary surgical procedures.

In order to obviate these errors in my department at the Research Clinic, I insist that each patient who complains of headache be required to fill out a two-page detailed questionnaire. There are two fairly large blank spaces in which the patient may even elaborate on his symptoms. This, at least, makes him alert to the fact that I expect him to give me full details. I impress on him at the first consultation that one can seldom "see" a headache—it must be diagnosed by the symptoms. The one exception is the instance in which one is fortunate enough to see a patient in an attack. Since the pain usually occurs at night this is seldom possible. Nearly all patients will say they have sinus trouble because of the nasal symptoms which occur in histaminic cephalalgia. None has an empyema of the sinuses; many have a dysfunction of the nasal mucosa with profuse secretion in the nose and nasopharynx. This, *per se,* is not the cause of the pain, but it does provide a localizing symptomatic starting point.

MANAGEMENT AND TREATMENT.

Someone has said, "If I wished to show a student the difficulties of medical practice, I should give him a headache to treat." May I add to this sagacious remark that the physician must be prepared to expect these "difficulties" and to adjust himself to them. He must be unwilling to accept defeat. He must insist on ample time for observation and stabilization of therapy. The physician positively must not accept patients and be responsible for results unless the patient can be observed for at least one month. If the patient's progress is not satisfactory, the physician must be alert and willing to change any or all the therapeutic procedures. In a number of instances I have changed the whole unfavorable outlook by restudying the hormonal status of the patient. I test the patient over a three-day period to determine the minimal effective dose of hormones. The physician must not accuse the patient of being psychotic because good results are not immediately obtained.

Fractional histamine is the most important part of the treatment and should be continued indefinitely, yet it does not by any means comprise the entire necessary therapeutic regime. The pain is produced by the vasodilating mechanism, with a disturbance of rhythm between vasodilation and vasoconstriction. Before the perplexing, dramatic and agonizing disease can be treated with any hope of success, certain fundamental physiologic and pharmacologic facts must be known and applied by the physician:

> Epinephrine constricts extracranial vessels; does not constrict intracranial vessels. It may be tried in doses of 1 cc. of a 1:100,000 solution, intravenously, to relieve the acute attack.
>
> DHE_{45} constricts extracranial and intracranial vessels and will relieve the acute attack in a few minutes. It must be given intravenously. Opiates will not give relief. A vasoconstrictor is the only effective remedy.
>
> Histamine dilates both intracranial and extracranial vessels, arterioles, venules and capillaries.
>
> Epinephrine and histamine are physiologic antagonists, except for their action on the intracranial vessels.

I know of no other disease that requires more intensive study and direction by the physician. I believe that there is a dysfunction of the autonomic nervous system, and probably a deficiency or imbalance of many of the body's most important physiologic processes. Horton[1] says, "There is no short cut in the treatment of headache, and the so-called sure cures usually prove disappointing." He advises histamine desensitization as follows: He gives subcutaneously twice daily—8 a.m. and 4 p.m.—Histamine Diphosphate 0.275 mg. per cc.

dosage to 0.15, 0.25, or 0.30 two (2) or three (3) times a day. We seldom find it necessary to go above the dosage just mentioned. It is our object to keep the dosage as small as possible. Be sure to inform us each time you return to the office, exactly how much pain you have had. When you go two weeks without severe pain, we usually reduce the dosage or increase the interval between doses. If you notice flushing of your face after taking Histamine please report this to us.

Steps for Filling Syringe:

Wipe off top of bottle with alcohol sponge or cotton. Pull back on the plunger of the syringe and inject some air into the bottle being sure to have the syringe and plunger securely fitted into the hand. After injecting the air, draw back more solution into the syringe than needed, holding the bottle up straight. This is necessary in order to get any air bubbles out of the syringe, back into the bottle, as you cannot possibly have the accurate dosage required when there is air in the syringe.

With the plunger on the exact mark of dosage, remove the syringe from the bottle. May we say again, PLEASE HOLD PLUNGER AND SYRINGE SECURELY.

Preparation of the Skin for Injection:

This is more easily given in the upper leg.

Wipe the area of injection with an alcohol sponge before giving yourself the injection.

With your free hand hold up on the flesh securely and stick the needle straight into the flesh, push the plunger down into the syringe, and then remove quickly, wiping the area once again with an alcohol sponge.

If DHE_{45} or other medication is to be given use the same technique in handling the syringe.

3. The patient cannot stay in bed with this type of head pain.
4. There is nasal stuffiness and profuse lacrimation, redness and swelling of the eye on the affected side.
5. The external and internal carotid arteries are both tender during the acute attack. Many patients find that compression over these areas will give some temporary relief.

This paper describes one of the most painful syndromes known. A wrong diagnosis is the rule, not the exception, even by most experienced physicians.

This presentation offers nothing that is new except the low dosage of histamine during the desensitization period, and minute detailed attention to deficiencies of all sorts, including hormones.

To Dr. Bayard T. Horton should go great honor from the medical profession all over the world for his splendid clinical research on the disease that bears his name—"Horton's Syndrome."

REFERENCES.

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1110 Professional Bldg.

were unconscious, but only twitching and inconstant minor convulsions were present. Death could be produced rapidly by the further injection of 0.2 cc. intravenously.

From the above results, cocaine would appear to be at least twice as toxic as hexylcaine.

Absorption from the tracheal mucosa was rapid in each case and, as has been mentioned, the recovery was moderately rapid when injection was discontinued.

Clinical experiments. Intranasal tests:

Following the procedure developed previously, the potency of hexylcaine was compared with that of cocaine by testing and comparing 5 per cent solutions of each drug when applied to the nasal mucosa of patients visiting the Out-patient Department of the Toronto General Hospital. Each patient was examined prior to testing, and particular attention was paid to the condition of the nasal mucosa of each individual. Only those patients with apparently normal mucous membrane were used. Eight men and four women, ranging in age from 18-60 were used.

The procedure was as follows: Half inch squares of blotting paper impregnated with constant amounts of 5 per cent cocaine and 5 per cent hexylcaine hydrochloride were inserted into the nose and applied to the nasal septum after first determining that normal sensation to pin prick was present. The cocaine was applied to one side and the hexylcaine to the other side of the nasal septum. Sensation was tested at one minute intervals until the patient declared that the sharp pin prick sensation was absent. This time was called the time of onset. The blotting paper was then removed and the time for return of normal sensation was noted and referred to as time of duration.

RESULTS—*See Table 1.*

No significant difference could be seen in times of onset with the two anesthetics but there was a constant difference in time of duration, hexylcaine being slightly longer than cocaine. This difference was from one to two minutes.

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TABLE 1.

POTENCY STUDIES
Solutions applied to nasal mucosa of septum

Cocaine 5%		Cyclaine 5%	
Onset in min.	Duration in min.	Onset in min.	Duration in min.
3	5	2	7
3	4	4	5
3	7	2	9
2½	2	2½	4
2	2	2	6
2½	6	2½	11
2½	5	2	8
1	8	1	10
2	6	2	9
2½	8	3	8
1	5	1	7
2	6	2	8

Clinical Trials. Endoscopic examination.

In the past, cocaine hydrochloride has been the anesthetic most often used for endoscopic procedure at the Toronto General Hospital, except for a one- to two-year period when tetracaine was used widely. The concentration of cocaine most often used was a 5 per cent solution. It was decided to try 5 per cent hexylcaine as the local anesthetic agent, using as a basis of comparison past experience with cocaine.

Twenty patients were used. The pre-operative sedative in each case was the same as that administered when cocaine was used (i.e. morphine 1/6 gr.-1/4 gr., atropine 1/150 gr., nembutal 1½ gr. The method adopted for applying the anesthetic was identical with that used with 5 per cent cocaine and was as follows: The pharynx was sprayed with approximately 2 cc. of the solution after which both piriform fossae and the supra glottic area were swabbed with cotton pledgets moistened with 5 per cent hexylcaine. Two cc. of solution was then dropped between the vocal cords into the trachea. The total amount used did not exceed 5 cc.

The amount of coughing observed during the endoscopic procedure was the main criterion for comparison. The results were tabulated as good (i.e. as good as the best results previously obtained with 5 per cent cocaine), fair, and poor. In 18 of the 20 cases epinephrine in a final concentration of 1:50,000 was used with the anesthetic solution. In two cases no epinephrine was used.

RESULTS.

Fourteen of the 18 cases showed good anesthesia. Four cases were classified as fair, but it was felt that these cases would have been only fair with any topical anesthetic. All four were seen to have excess mucus, generalized thickening of the mucous membrane, or generalized acute inflammation of the mucous membrane.

The two cases which received hexylcaine without epinephrine were similar in every respect to those who did receive epinephrine in the anesthetic solution.

No untoward reactions were noted. There was no evidence of twitching or convulsions, and there was no visible damage to the tracheal or bronchial mucosa.

It was felt that hexylcaine in a 5 per cent solution produced anesthesia as satisfactory as might be expected with 5 per cent cocaine.

Hydrolysis in human serum.

The hydrolysis of hexylcaine in human serum was observed over a period of time, using the Beckman quartz spectrophotometer. Moderately purified human serum esterase was prepared from outdated blood from a blood bank. Studies were done at room temperature and results with hexylcaine were compared with procaine hydrolysis performed in a similar manner. Detailed description of the method of studying the hydrolysis is omitted from this paper and the reader is referred to a paper by Kalow.[2]

It was found that hexlycaine was broken down slowly in human serum, four to eight times more slowly than procaine. In 100 minutes 3.5 micrograms of procaine HC1 as compared to 0.8 to 0.4 micrograms of hexylcaine HC1 were destroyed by the same preparation of human serum estarase. This finding contrasted with the observation made during toxicity studies with the experimental animal where it could be safely assumed that hexylcaine was broken down rapidly, allowing the animal to recover if the injections of the drug were interrupted for a few minutes. This finding serves to emphasize what is seen frequently, namely, that animal toxicity studies in particular cannot be safely applied to the human subject.

It is known that cocaine undergoes almost negligible hydrolysis in human serum, and the rate of hydrolysis of procaine is considered to be moderately rapid.

SUMMARY.

1. Toxicity studies using guinea pigs are presented and it is seen that cocaine is at least twice as toxic as hexlycaine.

2. Potency studies using humans are presented. Hexylcaine is found to produce anesthesia at least as prolonged as with an equal amount of cocaine.

Hexylcaine was found to be as efficient as cocaine in the production of local anesthesia *for the performance of endoscopic procedures.*

3. The hydrolysis of hexylcaine by human serum esterase was found to be four to eight times slower than hydrolysis of procaine.

CONCLUSIONS.

Hexylcaine appears to be as efficient a local anesthetic as cocaine for endoscopic procedures and appears to have a greater margin of safety than cocaine.

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METASTASIS OF MALIGNANT TUMORS TO THE LARYNX.

JOHN R. LOUGHEAD, M.D., Poplar Bluff, Mo.,
and
JAMES BUSHNELL, M.D., St. Louis, Mo.

The purpose of this article is to note the unusual occurrence of malignant tumors metastizing to the larynx. The lymphomata group are not included.

A review of the world literature from 1916 through 1951 reveals five authentic cases of tumor metastasis to the larynx.

CASE REPORTS.

Case 1: In 1924 Turner reported the first well documented case of a renal cell carcinoma metastizing to the larynx. A seventy-year-old white male was admitted to the hospital with a chief complaint of hoarseness of three months' duration. A smooth pale subglottic swelling directly beneath the right true cord was noted on examination. At autopsy this mass was found to be a metastasis from a renal cell carcinoma of the right kidney.[1]

Case 2: In 1931 Oppikoder reported the case of a fifty-year-old white male who entered the clinic with a chief complaint of dysphagia of two months' duration. A one centimeter, nodular, blue-red mass involving the left half of the epiglottis was found on examination. The tumor was locally excised and thought to be either a metastasis from a renal cell carcinoma, a thyroid carcinoma or a sarcoma. A review of the patient's history revealed that a left nephrectomy for a renal cell carcinoma had been done six years prior to the discovery of the epiglottic lesion. No further tumor spread was noted for one and one-half years following local removal of the epiglotte mass.[2]

Case 3: In 1941 Havens and Parkhill reported the case of a forty-year-old white male who entered the clinic with a chief complaint of throat irritation of one year's duration. A malignant appearing dark brown mass was seen arising from the right arytenoid cartilage. Pathological examination of the tumor revealed a diagnosis of malignant melanoma. Other widespread metastasis presumably from a primary skin lesion were present.[3]

Case 4: In 1951 Fisher and Odess reported the case of a sixty-three-year-old white female who entered the hospital with a chief complaint of hoarseness of eight months' duration. On examination a round, smooth, dark colored tumor involving the right vocal cord was seen.

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A malignant melanoma had been removed from the right shoulder three years previously. A laryngectomy was done, and a diagnosis of malignant melanoma of the larynx was made.[4]

Case 5: In 1952 one of us (J. R. L.) reported the case of a sixty-eight-year-old white male who entered the hospital wth a chief complaint of hoarseness of five months' duration. A greyish-pink, grape-like tumor 6 mm. in diameter was seen attached to the left true cord. This was removed and found to be a malignant melanoma. The probable primary lesion was found on the skin of the right shoulder. Other wide spread metastasis soon became apparent.[5]

Several cases of lesions involving the larynx which were presumably metastatic growths from renal cell carcinomas are present in the literature but were not proven by renal examination.[1,2,6,7]

DISCUSSION.

Two theories are generally accepted to account for the scarcity of tumor metastasis to certain regions:

(1) Some tissues present less favorable environment for tumor growth.

(2) Failure of tumor emboli to reach certain[8] regions in sufficient number for growth.

In support of the latter, Coman et al., in experimental work with sarcoma in rabbits, found that tumor metastasis would develop in any organ that received viable tumor emboli in sufficient numbers. Muscle metastasis developed readily after injection of emboli into the left heart chamber.[9]

Tumor emboli are probably arrested in the first capillary network encountered. Emboli liberated in the systemic veins are caught in the lungs, those entering the portal may lodge in the liver and those entering the pulmonary veins may presumably be deposited in any peripheral organ.[8]

Batson pointed out the possible role of the vertebral veins in the spread of metastasis by retrograde flow thus yielding systemic spread without passing through the lungs.[10]

The location of the larynx is such that it is probably reached by relatively few tumor emboli. Only those tumors which metastasize widely and profusely would presumably be encountered as metastatic to the larynx. Renal cell carcinoma and malignant melanoma fall into this category.

SUMMARY.

A review of the literature reveals two acceptable cases of metastasis of renal cell carcinoma to the larynx and three cases of metastasis of malignant melanoma to the larynx. It is believed that the scarcity of metastatic lesions involving the larynx is due to the scarcity of tumor emboli reaching the larynx.

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HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

January 1, 1954.

Acousticon Models A-17, A-180 and A-185.
Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 1, New York.

Auditone Models 11 and 15.
Manufacturer: Audio Co. of America, 5805 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67 and 70.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Symphonette; Beltone Mono-Pac Model M; Mono-Pac Model "Lyric"; Mono-Pac Model "Rhapsody."
Manufacturer: Beltone Hearing Aid Co., 2900 West 36th St., Chicago 32, Ill.

Cleartone Model 500; Model 700; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Model D-1; Dahlberg Junior Model D-2; Dahlberg Model D-3 Tru-Sonic; Dahlberg Model D-4 Tru-Sonic.
Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Fortiphone Models 19-LR; 20A; 21-C and 22.
Manufacturer: Fortiphone Limited, Fortiphone House, 247 Regent St., London W. 1, England.
Distributor: Anton Heilman, 75 Madison Ave., New York 16, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.
Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.
Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico Model J; Maico Top Secret Model L; Maico Maxitone.

Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis, Minn.

Micronic Model 303; Micronic Model "Mercury"; Micronic Star Model.

Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Classic Model T9; Microtone Model T10; Microtone Model T612.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 106 So. 7th St., Philadelphia 6, Pa.

Normatone Model C and Model D-53.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.

Distributor: Normatone Hearing Aid Co., 22 East 7th St., St. Paul (1), Minn.

Otarion Models B-15 and B-30; Otarion Models F-1, F-2 and F-3; Otarion Model G-2; Otarion Model G-3; Otarion Model H-1; Custom "5."

Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40. Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny-Mite); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Model 62 Starlet; Model 72; Model 82 (Zephyr).

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Distributor: Radioear Corp., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Silvertone Model H-16, J-92; Silvertone Model P-15.

Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.

Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99
Manufacturer Solo-Pak Electronics Corp., Linden St., Reading, Mass

Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966; Sonotone Model 977; Sonotone Model 988
Manufacturer Sonotone Corp, Elmsford, N Y

Televox Model E.
Manufacturer Televox Mfg Co, 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 953; Telex Model 1700.
Manufacturer Telex, Inc., Telex Park, St. Paul 1, Minn

Tonamic Model 50.
Manufacturer Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster; Model Cameo.
Manufacturer Tonemasters, Inc., 400 S Washington St., Peoria 2, Ill

Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.
Manufacturer Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2
Manufacturer Vacolite Co 3003 N Henderson St. Dallas 6 Tex.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal; Zenith "Regent."
Manufacturer Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices have vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TRANSISTOR HEARING AIDS ACCEPTED.

Acousticon Model A-300; 1 transistor, 2 tubes, and 2 batteries.
Manufacturer: Dictograph Products., Inc., 95-25 149th St., Jamaica 35, N. Y.

Audivox, Model 71; all transistor.
Manufacturer: Audivox, Inc., 123 Worcester St., Boston 18, Mass.

Maico Transist-Ear, Model O; 3 transistors and 1 battery.
Manufacturer: The Maico Company, Inc., 21 N. 3rd St., Minneapolis, 1.

Otarion Model C-15; 1 transistor, 2 tubes, and 2 batteries (A & B).
Manufacturer: Otarion, Inc., 4757 N. Ravenswood Ave., Chicago 40, Ill.

Sonotone Model 1010; 1 transistor, 2 tubes, and 2 batteries (A & B).
Manufacturer: Sonotone Corporation, Elmsford, N. Y.

Telex Model 954; 1 transistor, 2 tubes, and 2 batteries (A & B).
Manufacturer: Telex, Inc., Telex Park, St. Paul, 1.

Zenith Model Royal-T; 3 transistors, and 1 battery. Zenith Model Super Royal-T; 3 transistors, and 1 battery.
Manufacturer: Zenith Radio Corp., 5801 W. Dickens Ave., Chicago 39, Illinois.

SEMI PORTABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).
Manufacturer: A. M. Brooks Co., 1222 W. Washington Blvd., Los Angeles 7, Calif.

Aurex (Semi-Portable).
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.
Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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Southern Section Saturday, Jan. 16, 1954, Louisville Ky., Brown Hotel
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Secretary: Dr. F. Johnson Putney, 255 So. 17th St., Philadelphia (3) Pa.
Meeting: Statler Hotel, Boston, Mass. (Afternoons) May 25-26, 1954.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. Victor Alfaro.
Vice-Chairman: Dr. Irvin Feldman.
Secretary: Dr. Frasier Williams.
Treasurer: Dr. John Louzan.
Meetings are held on the third Tuesday of October, November, March and May, 7:00 P.M.
Place: Army and Navy Club, Washington, D. C.

THE LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. W. L. Hughes, Lamar Life Bldg., Jackson, Miss.
Vice-President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

OTOSCLEROSIS STUDY GROUP.

President: Theo. E. Walsh, 640 So. Kingshighway, St. Louis 10, Mo.
Secretary: Dr. Lawrence R. Boies, Med. Arts Bldg., Minneapolis 2, Minn.
Meeting: Waldorf-Astoria, New York City, Sept., 1954.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis, Ind.
President-Elect: Dr. Albert D. Ruedemann, 1633 David Whitney Bldg., Detroit 26, Mich.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2, N. Y.
Meeting: Waldorf-Astoria, New York City, Sept., 1954.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Justo M. Alonso, Montevideo.
Executive Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U. S. A.
Meeting: Fourth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
President: Dr. Ricardo Tapia Acuna, Mexico City.
Time and Place: Feb. 28 to Mar. 4, 1954, Mexico City.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Norris J. Heckel, Chicago, Ill.
President-Elect: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.
Meeting: Chicago, Ill., Sept. 22-24, 1954.

THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Peter N Pastore, Richmond, Va.
President-Elect: Dr. G. S. Fitz-Hugh, Charlottesville, Va.
Vice-President: Dr. H. L. Mitchell, Lexington, Va.
Secretary-Treasurer: Dr. L. B. Sheppard, 301 Medical Arts Bldg., Richmond, Va.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Orwyn H. Ellis, M.D.
Secretary-Treasurer: Harold Owens, M.D.
Chairman of Section on Ophthalmology: Robert A. Norene, M.D.
Secretary of Section on Ophthalmology: Sol Rome, M.D.
Chairman of Section on Otolaryngology: Leland R. House, M.D.
Secretary of Section on Otolaryngology: Max E. Pohlman, M.D.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:00 P.M., fourth Monday of each month from September to June, inclusive—Otolaryngology Section; 6:00 P.M., first Thursday of each month from September to June, inclusive—Ophthalmology Section.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Harry Nievert, 555 Park Ave., New York (21), N. Y.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York (16), N. Y.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. William Banks Anderson, Durham, N. Car.
Secretary and Treasurer: Dr. Geo. B. Ferguson, Durham, N. Car.
Meeting: Joint, with South Carolina Society of Ophthalmology and Otolaryngology, Durham, N. C., Nov. 4-6, 1954.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. David S. Asbill, Columbia, S. Car.
Vice-President: Dr. John McLean, Greenville, S. Car.
Secretary-Treasurer: Dr. Roderick Macdonald, Rock Hill, S. Car.
Meeting: Joint, with North Carolina Eye, Ear, Nose and Throat Society, Durham, N. C., Nov. 4-6, 1954.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.
Secretary-Treasurer: Dr. John F. Tolan, 3419 47th Ave., Seattle (5), Wash.
Meeting: Honolulu, 1954.

THE RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Chairman: Dr. Isaac H. Jones, 635 S. Westlake, Los Angeles, Calif.
Treasurer: Dr. Pierre Violé, 1930 Wilshire Blvd., Los Angeles, Calif.
Program Chairmen:
Otolaryngology: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.
Ophthalmology: Dr. Harold F. Whalman, 727 W. 7th St., Los Angeles, Calif.

Mid-Winter Clinical Convention annually the last two weeks in January at Los Angeles, Calif.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Chas. C. Grace, 145 King St., St. Augustine, Fla.
President-Elect: Dr. Jos. W. Taylor, 706 Franklin St., Tampa, Fla.
Secretary-Treasurer: Dr. Carl S. McLemore, 1217 Kuhl Ave., Orlando, Fla.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President: Dr. Harry P. Schenck.
Vice-President: Dr. William J. Hitschler.
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Secretary: Dr. John J. O'Keefe.
Historian: Dr. Herman B. Cohen.
Executive Committee: Dr. M. Valentine Miller, Dr. Charles E. Towson, Dr. Thomas F. Furlong, Dr. Benjamin H. Shuster, ex-officio.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Vice-Chairman: Dr. K. W. Cosgrove, 111 E. Capitol Ave., Little Rock, Ark.
Secretary: Dr. F. A. Holden, Medical Arts Bldg., Baltimore, Md.
Meeting:

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James K. Stewart, Wheeling, W. Va.
Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. C. Otrich, Belleville, Ill.
President-Elect: Dr. Phil R. McGrath, Peoria, Ill.
Secretary-Treasurer: Dr. Alfred G. Schultz, Jacksonville, Ill.

CANADIAN OTOLARYNGOLOGICAL SOCIETY
SOCIETE CANADIENNE D'OTOLARYNGOLOGIE

President: Dr. D. E. S. Wishart, 170 St. George St., Toronto, Ontario.
Secretary: Dr. W. Ross Wright, 361 Regent St., Fredericton, N. B.
Place:
Time:

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Oscar Marchman, Jr., Dallas, Texas.
Secretary-Treasurer: Dr. Morris F. Waldman, Dallas, Texas.

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDICO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Salvador Mixco Pinto.
Secretary: Dr. Daniel Alfredo Alfaro.
Treasurer: Dr. Antonio Pineda M.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

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Vice-President: Dr. M. Gonzalez Ulloa, Mexico, D. F.
Secretary: Dr. Juan de Dios Peza, Mexico, D. F.

FEDERACION ARGENTINA, DE SOCIEDADES DE OTORRINOLARINGOLOGIA.

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Sub-Secretario del Exterior: Dr. Oreste E. Bergaglio.
Secretario del Interior: Dr. Eduardo Casterán.
Sub-Secretario del Interior: Dr.Atilio Viale del Carril.
Secretario Tesorero: Dr. Vicente Carri.
Sub-Secretario Tesorero: Dr. José D. Suberviola.

ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

Presidente: Dr. J. Abello.
Vice-Presdente: Dr. Luis Suñe Medan.
Secretario: Dr. Jorge Perelló, 319 Provenza, Barcelona.
Vice-Secretario: Dr. A. Pinart.
Vocal: Dr. J. M. Ferrando.

SOCIEDAD NACIONAL DE CIRUGIA OF CUBA.

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Vicepresidente: Dr. César Cabrera Calderin.
Secretario: Dr. José Xirau.
Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Andre Soulas, Paris, France.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa. U. S. A.
Meeting: 3rd International Congress of Broncho-Esophagology.
Time and Place: September or October, 1954, Lisbon, Portugal.

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER — CAMPINAS.

President: Dr. Heitor Nascimento.
First Secretary: Dr. Roberto Barbosa.
Second Secretary: Dr. Roberto Franco do Amaral.
Librarian-Treasurer: Dr. Leoncio de Souza Queiroz.
Editors for the Archives of the Society: Dr. Guedes de Melo Filho, Dr. Penido Burnier and Dr. Gabriel Porto.

SOCIEDAD CUBANA DE OTO-LARINGOLOGIA.

President: Dr. Reinaldo de Villiers.
Vice-President: Dr. Jorge de Cárdenas.
Secretary: Dr. Pablo Hernandez.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA.

Presidente: Dr. Aldo Remorino.
Vice-Presidente: Dr. Luis E. Olsen.
Secretario: Dr. Eugenio Romero Díaz.
Tesorero: Dr. Juan Manuel Pradales.
Vocales: Dr. Osvaldo Suárez, Dr. Nondier Asís R., Dr. Jorge Bergallo Yofre.

BUENOS AIRES CLUB OTORINOLARINGOLOGICO.

Presidente: Dr. Alberto P. Haedo.
Vice-Presidente: Dr. V. R. Carri.
Secretario: Dr. Renoto Segre.
Pro-Secretario: Dr. Carlos A. Gutierrez.
Tesorero: Dr. J. M. Tato.
Pro-Tesorero: Dr. Norberto Von Soubiron.

SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLOMBIA).

Presidente: Dr. Alfonso Tribin P.
Secretario: Dr. Felix E. Lozano.
Tesorero: Dr. Mario Arenas A.

SOCIEDAD ESPANOLA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. D. Adolfo Hinojar Pons.
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Secretario General: Dr. D. Francisco Marañés.
Tesorero: Dr. D. Ernesto Alonso Ferrer.

ASOCIACION DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DE GUATEMALA

Presidente: Dr. Julio Quevedo, 15 Calle Oriente No. 5.
First Vice-Presidente: Dr. Héctor Cruz, 3a Avenida Sur No. 72.
Second Vice-Presidente: Dr. José Luis Escamilla, 5a Calle Poniente No. 48.
Secretario-Tesorero: Dr. Horace Polanco, 13 Calle Poniente No. 9-D.

FIRST CENTRAL AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY.

President: Dr. Victor M. Noubleau, San Salvador.
Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

THIRD LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Franze Conde Jahn, Caracas.
Secretary: Dr. Victorino Marquez Reveron, Caracas.
Meeting: Caracas, Venezuela, Feb. 21-25, 1954.

BOOK REVIEW.

Respiratory Diseases and Allergy. New Method of Approach. By Josef S. Smul, M.D., Fellow, National Gastro-Ent. Association; Formerly Vice President, Manhattan Roentgen Ray Society; Association of Gastro-Ent; Beth David Hospital; Clinical Assistant, Physicians Beth Israel Hospital. Eighty Pages with Index. New York: Medical Library Company, 232 East 15th Str. 1953. Price $2.75.

A note book is of not much value to anyone other than the author. This is a note book. It contains nothing new except some terms such as Respirallergy, Laryngallergy, etc., without which we would be much better off!

The book discusses respiratory diseases in some 72 pages; has an Index and one reference! T. E. W.

ANNOUNCEMENT.

The Annual Convention of the Oklahoma City Academy of Ophthalmology and Otolaryngology with the Postgraduate Department of the University of Oklahoma Medical School will be held March 26, 1954. Guest speakers will be Paul A. Chandler, M.D., Harvard University, Ophthalmologist, Boston, Massachusetts, and Theo. E. Walsh, M.D., Washington University, Otolaryngologist, St. Louis, Missouri.

Registration fee of $12.00 includes registration, social hour, and dinner. The program will include question and answer periods.

1954 CONGRESS OF THE SOCIETA ITALIANA DI OTORINOLARINGOIATRIA.

For the next Congress of the Societá Italiana di Otorinolaringoiatria in 1954 the following report subject has been chosen: "Congenital Malformations in the field of Oto-rhinolaryngology."

The authors who have published their researches on this subject are kindly asked to communicate the titles to Dott. Tomaso Marullo, Clinica Otorinolaringoiatria, Universitá, Roma, or, if possible, to send him a presentation copy.

BRONCHOESOPHAGOLOGY COURSE.

The next Bronchoesophagology Course to be given by the University of Illinois College of Medicine is scheduled for the period, March 22 through April 3, 1954. The course is under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

PROCEEDINGS OF BRITISH ASSOCIATION OF OTOLARYNGOLOGISTS.

The British Association of Otolaryngologists has copies of the Proceedings of the 4th International Congress of Otorhinolaryngology for sale at Five Guineas each.

These papers are numerous and the publication is well produced.

Application for copies should be made to The Secretary, British Medical Journal, B.M.A. House, Tavistock Square, London, W.C.1.

KENFIELD MEMORIAL AWARD.

Competition for the Kenfield Memorial Scholarship, awarded annually by the American Hearing Society to a prospective teacher of lipreading, will open March 1. Application blanks may be obtained by writing to the society's national headquarters, 817 - 14th St., N.W., Washington 5, D. C.

Deadline for returning completed applications is May 1. They are to be mailed to Mrs. Eleanor C. Ronnei, care of the New York League for the Hard of Hearing, 480 Lexington Ave., New York 17, N. Y. The winner will be announced during National Hearing Week, May 2 - 8.

Winner of the annual award is entitled to take a teacher training course in lipreading from any school or university in the United States acceptable to the Teachers Committee. The scholarship is to be used within one year from the date of award.

A satisfactory applicant for the award must be a well adjusted individual with a pleasing personality, legible lips, good speech and voice and no unpleasant mannerisms. Graduation from college with a major in education, psychology and/or speech is a requirement.

Rules for competition state that an applicant shall plan to teach lipreading with or without other types of speech or hearing therapy.

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TEN YEARS' EXPERIENCE IN THE USE OF TANTALUM IN FRONTAL SINUS SURGERY.*†

Robert L. Goodale, M.D.,
Boston, Mass.

The purpose of this paper is to report on cases which I presented at a meeting of the New England Otolaryngological Society in Boston, May 23, 1945,[1] and to describe some of the problems which have occurred in the use of tantalum foil as a method of maintaining drainage from the frontal sinuses after the Lynch operation.

In a paper published in 1942,[2] the author analyzed the causes for failure in frontal sinus surgery. Of 190 cases operated upon at the Massachusetts Eye and Ear Infirmary between 1931 and 1942, 123 were done by the Lynch technique; and of these 30.18 per cent required reoperation at a later date.

At the second operation the causes for failure were discovered to be the following: *1.* obstruction of the naso-frontal passage by adhesions; *2.* remnants of frontal sinus floor which later gave rise to undrained, infected pockets in the frontal sinus; *3.* incomplete ethmoidectomy, especially failure to remove orbital extensions of the ethmoid.

It was apparent that the wide opening provided by the Lynch operation from the frontal sinus into the nose did not

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* Read at a meeting of the Eastern Section of the American Laryngological, Rhinological and Otological Society, Inc., January 8, 1954, New York, N. Y.

† From the Department of Otolaryngology, Massachusetts Eye and Ear Infirmary, Boston, Mass.

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remain patent because of a tendency for the orbital contents to push the orbital periosteum toward the midline, where it came in contact with the medial wall. If this area had been traumatized it was inevitable that granulation tissue should form, and as all granulation tissue eventually is transformed into scar tissue it was obvious that this passage would become obliterated.

The technique which the author adopted was suggested by consideration of this surgical-pathological situation. If only the two surfaces could be kept apart by an inert substance, the granulation tissue of one surface would not have a chance to attach itself to the opposite surface, and the passage would then remain patent although narrowed in some cases. In searching for such a material, the metal tantalum was chosen because it proved to be inert in animal tissue and could easily be cut and fitted to the area to be covered at the time of operation. This was based on the work of Pudenz,[3] who had used tantalum plates in cranial defects in animals. He concluded that tantalum is a satisfactory material for repair of cranial defects as it has the desirable qualities of being noncorrosive, is inert in tissue, is non-absorbable, with an absence of toxic ingredients, and malleability.

The material for the cases reported in 1945 was a tantalum foil 1.5 cm. wide, 4 cm. long, and .00025 inches in thickness; sutured to the orbital periosteum by .003 inch tantalum wire.

During the past 10 years a total of 20 such cases have been performed, either by the author or under his supervision. This would seem to be a very small series, but it is to be noted that this technique is recommended only for such cases where it is definitely unlikely that the naso-frontal passage will remain patent after a Lynch operation. It is not in any sense a routine procedure for all cases. It should also be noted that during the past 10 years we have had the antibiotic drugs to control sinus infection. It is my belief that this accounts for a considerable decrease in the number of cases which we have had at Massachusetts Eye and Ear Infirmary during this period. I herewith present a follow-up on the cases which were presented in the paper in 1945.

Case 1. "A man, 42 years of age, entered the Nose and Throat Service at Massachusetts Eye and Ear Infirmary in September, 1943. Preoperative X-ray films showed a mucocele of the right frontal sinus with marked sclerosis about both frontal sinuses and a perforation through the anterior wall of the right frontal sinus. On October 28, 1943, a radical fronto-ethmoidectomy was done, and the lining of the frontal sinus together with the mucocele was removed. As the X ray showed, there was a small perforation of the anterior wall near the supraorbital ridge. A strip of tantalum foil 1.5 cm. wide and 4 cm. long and .00025 inch in thickness was sutured to the orbital periosteum at about the position of the pulley (see Fig. 1). The foil was then carefully placed so that it was in contact with the orbital periosteum and extended medially and downward into the upper part of the nasal fossa. The suture material was .003 tantalum wire. The patient has tolerated this metal very well. There have been a few attacks of acute rhinitis, but the naso-frontal passage has remained patent and there has been no recurrence of the mucocele."

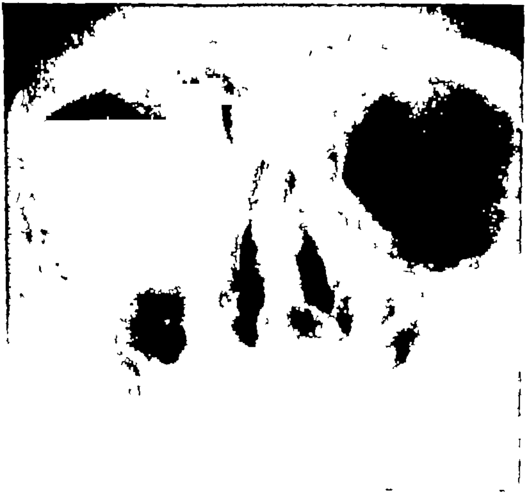

Fig. 1. Roentgenogram showing tantalum foil in place. This foil is attached at the upper end by tantalum wire to the orbital periosteum. The irregular outline is due to the fact that the sheet of tantalum is seen edge on. The foil extends downward through the area formerly occupied by the anterior ethmoid cells into the upper part of the nasal fossa.

This patient reported to me again, at my request, in October, 1953. I had not heard from him for five years and naturally wondered whether he had had further difficulty. I was pleasantly surprised when he gave a history of having had no trouble whatsoever with his frontal sinus since the operation. The tantalum was still in place; his nose was clean with no secretion. The only symptom he mentioned was that when he had occasional head colds his eyelids became slightly reddened, but this would last for only two or three days.

Case 2. "This was a girl, aged 15, who had had multiple left frontal sinus operations dating from March, 1942. Following each operation I attempted to keep the naso-frontal passage open by dilating the duct with a naso-frontal probe. In spite of repeated attempts at regular intervals, the passage narrowed until it had completely disappeared. On March 14, 1945, I reoperated and found that there was complete obstruction to drainage due to scar tissue and regeneration of bone near the nasal spine. The obstructing tissue was removed and tantalum foil was employed as in Case 1."

This patient has been under my observation since the first operation; she has had no more difficulty with her frontal sinus and the passage is open. Her only nasal difficulty since the operation was due to a nasal obstruction in the opposite nostril, which I corrected by a sub-mucous resection. She has had no recurrent infection in the frontal sinus.

Case 3. "This case illustrates the same difficulty. This was a patient who was referred to me for revisions of a right frontal sinus Lynch operation. The patient had been operated on for a fistula in an infected right antrum and for right chronic-fronto-ethmoiditis. The antrum had cleared up very satisfactorily, but the frontal sinus became blocked off by scar tissue. On May 11, 1945, I reopened the right frontal sinus through a Lynch incision. The adhesions were extremely dense and sprang from the region of the frontal spine to the orbital periosteum. The ethmoid was found to be completely exenterated by a previous operation. The sphenoid was entered easily and appeared to be in good condition. There was a little more bone to be taken off the ascending process. The frontal sinus contained mucopus and markedly thickened, infected mucous membrane. The floor had been well removed previously. I excised a great deal of organized scar tissue from the orbital periosteum and with a curette smoothed off some rough bone near the frontal spine. Above and below the frontal spine there seemed to be normal mucous membrane, which I hope will grow across the bare bone. Tantalum foil was used in this case with the same technique. The patient has had no reaction to the foil, and the frontal sinus has remained open."

About two years later the foil was removed, as the frontal sinus was open and the patient was symptom free. At last report he had had no further difficulty with his sinus.

Case 4. "This patient was a man, aged 64, who had suffered from right pansinusitis for many years. During the past winter, (1944), his nostrils had become completely blocked with nasal polypi, and had developed a persistent right frontal headache. In this case a right fronto-ethmoidectomy was performed. A slightly heavier tantalum foil, .0005 inch thick, was used (see Fig 2). His right antrum was not operated upon at this time It has remained quiescent since the operation He has a very clear, wide-open naso-frontal passage through which a probe passes easily into the frontal sinus."

This patient has been under my observation and was last seen at Massachusetts Eye and Ear Infirmary in October, 1953. An X-ray taken postoperatively is worth showing as it illustrates the position of the tantalum foil.

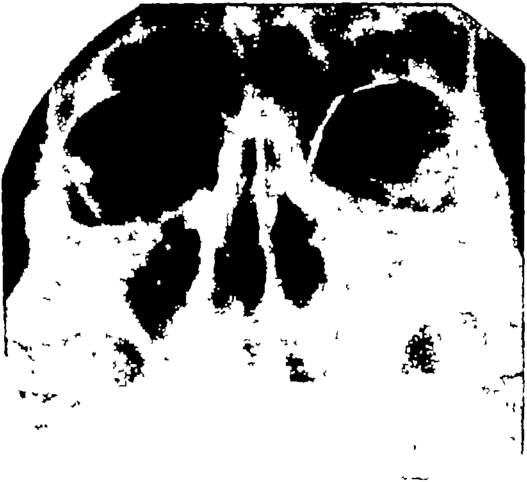

Fig 2 The tantalum can be seen approximately in the position of the floor of the right frontal sinus. In this case one suture was placed near the upper end of the sheet of foil and another suture at a point about 1 cm. from the lower end which lies in the upper part of the nasal fossa

In all these cases the tantalum foil was tolerated without discomfort; in fact, even when touching the foil with a probe the patient experienced no pain or discomfort and actually seemed to be unaware of its presence in the nose. The naso-frontal passages have remained clear in every case where the tantalum foil has remained in the proper position; that is to say, between the orbital periosteum and the midline in the region of the frontal spine.

In all, there have been 20 cases in which I have had a chance to evaluate the results. I would like to present some of the defects and errors in this technique: *1.* error in position of the tantalum foil; *2.* removal of the tantalum foil under a mistaken idea that it is a foreign body and, therefore, may block the frontal sinus; *3.* displacement of the foil by injudicious probing so that it no longer remains at the proper level in the nose.

To illustrate the first error there are two cases: One, (P. R.), was operated upon July 13, 1945. This patient had pansinusitis of severe, chronic, hyperplastic type in which tantalum was sutured into both frontal sinuses. On the left side the tantalum remained in place satisfactorily; on the right the anterior edge of the tantalum slipped into the line of incision so that it prevented proper union of the skin edges. A fistula formed in this area which necessitated the removal of the tantalum foil (Feb. 15, 1946); however, in this case the tantalum had remained in place for seven months. By that time epithelialization had occurred, which prevented adhesions from forming.

The second was a private case in which I assisted another surgeon. Here again the foil slipped forward into the incision and had to be removed. In other words, the very properties of tantalum foil which recommend it for use in the nasofrontal area interfere with the proper closure of the incision. For this reason, I recommend placing the tantalum foil well within the frontal sinus, and if necessary closing the wound in two layers in such a way that the deep suture lies external to the anterior edge of the foil. In this way it will prevent the slipping of the material. It is also possible as in Case 4, to suture the foil at the upper and lower end, thus anchoring it securely to the periosteum.

To illustrate the second problem, that of removal of the foil on the theory that it is a foreign body, I describe the following case: This was an 18-year-old boy, (T. K.), who had had repeated frontal sinus infection eventually leading to a chronic empyema of the sinuses. One month after the operation he picked up an acute nasal infection. At that time he was living in a distant city, and the surgeon in attendance removed the tantalum foil. The frontal sinus immediately became blocked with granulation tissue, so he presented me with the problem of further radical surgery. It seemed to me that in his case, if he again became reinfected, the whole story might be repeated; so I decided to obliterate his sinus completely and have a plastic repair of the defect done at a later date. On June 8, 1947, I obliterated the frontal sinus, and nine months later a plastic operation was performed by Dr. Edgar M. Holmes with a very satisfactory cosmetic result.

To illustrate the third problem, that of the foil becoming displaced by post-operative instrumentation, I quote the following case: This woman, age 51, entered Massachusetts Eye and Ear Infirmary with a diagnosis of right pansinusitis. Lynch operations were performed on October 30, 1950, and again on April 3, 1951, and as there was a strong likelihood that adhesions would form in the naso-frontal area, tantalum was sutured in the usual manner to the orbital periosteum. She returned again in November, 1951; at that time X-ray showed that the tantalum foil had been pushed back into the frontal sinus. A probe could not be passed into the sinus, as it was blocked by dense scar tissue. This patient was operated upon November 13, 1951, at which time an obliterative procedure was undertaken. The tantalum foil was removed and the patient had no further trouble in her sinus; however, the post-operative defect was a psychological hazard, so that on December 23, 1953, a bone graft was inserted to restore the contours of the forehead.

This brings up the question of the procedure which should be followed in difficult cases where reinfection is likely. As I have said before, the use of tantalum foil is not to be regarded as a routine procedure. If, in the opinion of the surgeon, an obliterative procedure such as I have just described offers a better outlook for the patient, it should be done; but where there is still a chance that the Lynch operation will succeed, and where the problem is one of maintaining naso-frontal drainage, I have found that tantalum foil has been most satisfactory.

I might mention three cases in which I was disappointed, not because of the tantalum foil, but because of other circumstances which occurred:

Case 1 was operated upon at Massachusetts Eye and Ear Infirmary by another member of the staff and seemed to be a fairly typical case for the use of tantalum. The surgeon removed a portion of the middle turbinate and sent it for pathological examination in a routine manner. He was not suspicious of malignancy which, however, proved to be the case. When the pathology report became available it was quite obvious that more surgery was indicated than originally contemplated; and, therefore, the tantalum was removed and further surgical procedures were undertaken.

Case 2 was a man with an alcoholic history. The surgeon who operated upon him was aware of this and thought that the pain which the patient experienced was of sinus origin, due to a pan-sinusitis. The Lynch operation was performed, and because of adhesions which made it difficult to maintain adequate drainage, tantalum was used. The pains continued, which later appeared to be due to alcoholic neuritis rather than to sinusitis.

Case 3 was a man who had a profound depression. In his case, although there was a severe sinus infection which was entirely cleared up by surgery, the depression became more marked. It was the opinion of the neuro-surgical service that the only relief for this patient was intra-cranial surgery, but the patient did not consent to this.

None of these three unfortunate patients had any complication from the use of tantalum.

SUMMARY.

In conclusion I again stress the fact that the use of any metal such as tantalum in frontal sinus surgery should be considered, not as a routine procedure, but rather in selected cases where it is difficult to maintain drainage from the sinus into the nose because of the tendency of scar tissue to form in this region. In my experience, when the tantalum has remained in the proper position, this has been successful. As in all surgery the proper selection of cases for any surgical procedure and the post-operative follow-up care is of great importance. I believe that in these cases, where it has been possible to keep track of the patients over a period of years, this method has proved successful.

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CONTACT ULCERS OF THE LARYNX.[*][†]

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(By Invitation).

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The clinical and pathological entity of contact ulcer of the larynx has been known to the laryngologist for many years. Jackson,[1] who gave the name to the condition in 1928, collected 127 cases over a period of 40 years. This would indicate his familiarity with this disease as far back as 1888.

This affection of the larynx is not common. In a period of 16 years, the writer has seen only 19 patients with nonspecific contact ulcers of the larynx. A study of this group has been made and is presented below. Particular importance is given to the value of vocal re-education as described by Peacher and Holinger,[2] for the treatment of these lesions.

By definition, contact ulcer is the superficial ulceration which may occur when one vocal process of any arytenoid cartilage comes in contact with the other vocal process. The ulceration may be unilateral or bilateral. The cause of the ulceration is trauma.

The traumatic factors have been described in detail by Jackson and Jackson,[3] and Jackson.[4] They have indicated that the trauma is brought about by a hammer and anvil type of mechanism; that is, the impact of the cartilaginous vocal processes against each other. The constant hammering is the cause of the ulceration.

Vocal abuse unquestionably adds a traumatic factor to the vocal process of the arytenoids. In certain individuals, who misuse their voices, the tips of these processes may bear a real strain on phonation. The muco-perichondrial covering of the tips is anatomically very thin, and it can be denuded when

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traumatized with consequent ulceration. This is a satisfactory explanation for the development of this nonspecific type of ulceration called a contact ulcer.

There are other causes of ulceration of the tips of the vocal processes. One of the sequelae of endotracheal anesthesia may be ulceration with granuloma formation in this location. The latter has the typical appearance of contact ulcer of the larynx; however, the etiological factor, as well as the treatment and prognosis, are different. The laryngeal lesions which may occur after endotracheal anesthesia offer no real diagnostic or therapeutic problem. Vocal rest, or vocal rest plus removal of the granulomas which may develop, usually effects a cure.

This is not the case with nonspecific contact ulcers. These have a different etiology and, therefore, the treatment and prognosis are different.

Johansen and Kaier,[5] made a thorough study of 21 patients with contact ulcers of the larynx. An autopsy was performed on 16 patients. There were 17 patients with pulmonary tuberculosis, in 14 of whom the ulceration was proved to be tuberculous, and in the remaining three, it was dubious. Typical nonspecific contact ulcers were demonstrated in four patients who had no pulmonary tuberculosis.

The work of Johansen and Kaier shows the importance of ruling out tuberculosis as a cause of contact ulcers.

The other important lesion which must be kept in mind is carcinoma. In my group of 19 patients, there was one case of proven squamous cell carcinoma. The patient had a superficial whitish ulcer over the tip of the right vocal process. A biopsy was taken and the diagnosis of carcinoma was established. X-ray therapy was given and the patient has remained well with an excellent voice for six years.

The pathological findings[6] in contact ulcer is that of a chronic nonspecific inflammation. The tissue examined may consist of new granulation tissue or fibrous inflammatory tissue, with or without cartilage in the bed of the ulcer.

SYMPTOMATOLOGY.

Contact ulcers usually develop in a patient who is not aware of any local or general illness. He has a sense of well being except for local symptoms to be discussed below. Occasionally, a history of a severe cold with paroxysmal coughing may be obtained. Also a patient may refer to overuse of his voice as a factor in the disease.

The most constant symptom is that of a sharp sticking pain in the region of the superior cornu of the thyroid cartilage on one side. The pain is aggravated by swallowing, and may be referred to the ear on the same side as the ulcer. It is very annoying and may be accompanied by a slight degree of hoarseness, cough and clearing of the throat. The patient is usually apprehensive because of the widespread publicity that has been given to the symptoms of cancer of the throat. As a result, he is apt to present himself for examination early in the course of the disease.

DIAGNOSIS.

The diagnosis of nonspecific contact ulcer of the larynx is not difficult to establish. Local symptoms are present and the patient will usually seek the advice of a physician, who refers him to the laryngologist. Occasionally, due to a lack of knowledge on the part of the examining physician, the diagnosis may be overlooked. As many as five different specialists examined one of the patients in this series and two specialists did not make the proper diagnosis.

By means of mirror laryngoscopy, typical lesions at the tip of one or both vocal processes are easily recognized. Ulcerations with or without granuloma are characteristic. It must be kept in mind that other specific lesions can produce ulceration or smooth-appearing projections in the posterior part of the larynx. Tuberculosis and carcinoma have especially to be kept in mind. The sequelae of endotracheal anesthesia must be ruled out. All patients with ulceration in this region should have an X-ray study of the chest and a biopsy before the diagnosis is established.

TREATMENT.

Before the work of Peacher and Holinger[3] in 1947, there seemed to be no definite plan of therapy. The role of vocal abuse as a cause of these lesions was known, but no one had investigated the importance of vocal re-education in the treatment of the condition. Patients were placed on long periods of absolute vocal rest. Laryngeal examination, if accompanied by a biopsy, frequently aggravated the condition. Cauterization or excision of the ulcers followed by vocal rest left much to be desired.

After a regime of absolute vocal rest, the ulcers would usually heal, only to be followed by a prompt recurrence when phonation was permitted.

Vocal re-education has been a definite step forward. After the diagnosis has been established, speech therapy is begun in the presence of the ulceration. For the patient this is most important economically, as he does not lose any time from work because of vocal rest. For the laryngologist, the opportunity to observe his patient recover from a very annoying nonspecific disease is gratifying. There is much less tendency for the lesion to recur if vocal re-education is successful.

CLINICAL OBSERVATION.

There were 19 patients observed, all of whom were males. The youngest was 25 and the oldest was 57. Occupation did not appear to be important.

Before 1947, 12 patients were seen, and the treatment after the diagnosis was established consisted of a regime of absolute vocal rest and occasional local excision of granulation tissue and gentle cauterization of the base of the ulcer. In only one instance was an attempt made to remove the tip of the vocal process. This was followed by almost disastrous results, and the case will be reported below. In this group of 12 patients who were treated in this manner, as far as can be determined, most of the patients did well. As a rule none was worse and all showed a good response to vocal rest. The effect of local therapy to the lesion was doubtful.

Many of the patients became disturbed because of the long time which was necessary to accomplish a good result. These sought help elsewhere, and not without the blessing of the doctor, because he had nothing more active to offer them.

Since 1947, seven patients have been seen and given the benefit of speech therapy. All have had a good result from vocal re-education. In three instances the patients were at the transition period and had been on vocal rest with or without local therapy to the lesion. After Peacher and Holinger's report, vocal re-education was started on these three patients with an excellent result.

It takes a well qualified speech teacher to give the type of therapy required in this laryngeal disorder; however, every qualified teacher will not necessarily secure a good result with every patient. There is a strong psychological factor in this type of therapy, and some patients did poorly with one teacher but responded well with another teacher; therefore, vocal re-education should not be condemned if it fails. A change in teachers may be all that is necessary in order to effect a cure. The average time required for a good result to become evident is about two months. There was one instance of recurrence among seven patients treated in this way, and it was brief.

With the advent of vocal re-education, the laryngologist and the patient with contact ulcer are both more tolerant of each other.

CASE REPORT.

W. M., white, male, age 48, was examined on September 27, 1946, and found to have an ulcer on the tip of the right vocal process. A direct laryngoscopy was performed and biopsy taken. A nonspecific chronic inflammation was reported. He was placed on absolute vocal rest, but a granuloma slowly formed in the area of ulceration. Three months later, in January, 1947, it was necessary to remove the granuloma from the region of the vocal process of the right arytenoid. He did not show any improvement and formed another granuloma in the same location. This was removed in April, 1947. He was again placed on vocal rest but failed to improve. In July, 1947, the tip of the right vocal process was excised along with the granuloma. In spite of vocal rest, a granuloma reformed and increased in size to such a degree that it was attached to the posterior two-thirds of the right vocal cord and caused some respiratory difficulty. It appeared that a tracheotomy might become necessary. It was evident that the granuloma was infected, and in desperation the patient was referred for X-ray therapy. He was given six X-ray treatments of 150 Roentgen units each making a total of 900 units over a period of two weeks. The granulation tissue promptly receded and decreased in size. At this time, Peacher and Hollinger's report came out. It was given careful study and the patient was referred to a speech therapist. Within a period of three months, his larynx healed and he has remained well without a recurrence to date.

SUMMARY.

Nonspecific contact ulcers of the larynx are not common. The important cause of the lesions is trauma, due to vocal abuse.

Differential diagnosis is based on the patient's history, the findings by indirect and direct laryngoscopy, X-ray study of the chest, and biopsy.

Treatment of nonspecific contact ulcers of the larynx should always include vocal re-education.

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THE OTOLOGIST'S ROLE IN THE INDUSTRIAL HEARING CONSERVATION PROGRAM.*

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Otologists in increasing numbers are enlisting in the struggle to mitigate the hazards of industrial noise and to conserve the hearing of exposed workers. Much has been written and said about occupational hearing loss and the conservation programs used in industry. It is commonly agreed that such programs, to be successful, require the cooperation of management and workers, health and safety personnel, and that a "team approach" is indicated.

Not only does the present writer endorse this opinion, but he would also seek to promote such teamwork through a candid exposition of the contribution which interested ear physicians can and should make to the properly conducted hearing conservation program. His observations and recommendations are based upon personal experience with hearing loss claims brought before the Wisconsin Industrial Commission, and with hearing conservation programs now in progress in the Wisconsin area.

The otologist who has interested himself in the many facets of occupational hearing loss is well suited to play a key role in the industrial hearing conservation program. As a result of his special training in the anatomy and function of the hearing mechanism, and his practical experience in the diagnosis and treatment of various hearing defects, he has acquired the skills needed for the proper management of a sound and purposeful program. He can offer realistic advice on such aspects of the industrial noise hazard as the following:

1. *On the "tailoring" of a Practical Program of Hearing Conservation to Meet the Needs of a Particular Plant.* It should never be overlooked that plants vary in their size,

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operation, production rates and acoustical characteristics, as well as in the number of workers. This means that each plant will have a "noise problem" which differs from its neighbor's or from plants which produce the same type of goods; therefore, the factors mentioned above, as well as others, such as the frequencies present in shop noises should be carefully considered. The program should neither be too costly nor require the worker to absent himself too long from productive work. Both workers and management must be instructed as to the need for and the purposes of the various procedures undertaken, if their cooperation is to be gained.

2. *On the Selection of a Suitable Room for the Conducting of Hearing Tests.* A room should be selected which is not too far from working areas, but which has, or can be treated to produce an ambient noise level low enough to permit the performance of practical hearing tests. A room with an overall noise level below 50 decibels is preferred but is not always obtainable in the industrial setting.

3. *On the Selection of the Audiometer and the Checking of its Performance.* In almost every plant a simple, but properly calibrated, and maintained, pure tone audiometer will meet all the needs for testing the hearing of workers in the plant. The accurate measurement of speech reception and discrimination tests, as well as the determination of bone conducting thresholds, requires optimum testing conditions and should be performed in the otologist's test rooms.

4. *On the Instruction and Guidance of Plant Personnel Conducting the Tests.* Experience has shown that the two most important requisites for the individual who is to perform the hearing tests are: *skill* and *interest.* Skill can be acquired through supervised practice, and interest can be stimulated by full instruction in the "whys and wherefores" of hearing and its measurement in the industrial setting.

5. *On the Evaluation of Pre-employment and Periodic Hearing Tests.* The interpretation of audiograms is a function best left to the medical man, as the one best prepared by training and experience to synthesize the medical and industrial histories with the results of hearing tests and otological examinations.

6. *On the Placement of Workers with Impaired Hearing.* This function is of particular importance where workers reveal significant or profound hearing losses at the time of hiring. It is essential that such workers be carefully examined and placed, after thorough hearing studies. Careful diagnostic differentiation between conductive, perceptive and mixed types of hearing losses also are of crucial importance in placing workers. The predominantly conductive deafness acts as "built-in ear plugs," while the predominantly perceptively deaf require careful watching less they be irreversibly aggravated.

7. *On the Selection, Fitting and Use of Ear Protective Devices.* Considering that any protective device to be inserted into the ear canal is essentially a foreign body, it becomes obvious that the supervision of this aspect of the hearing conservation program should be in the hands of medical personnel. It is imperative, too, that all necessary hygienic measures be taken.

8. *On the Susceptibility of Individual Workers to Occupation Hearing Loss.* Recently proposed test procedures suggest the feasibility of determining the susceptibility of individual workers to "noise deafness." There are differences, however, between the several proposed methods as to their reliability and time requirements.

9. *On the Detection of Willful Exaggeration of Either Hearing Ability, or Hearing Loss, by Workers.* Occasionally cases are encountered where a worker with appreciable loss, whose hearing has been tested several times in the past, will use his familiarity with the tests to "pass" a perfunctory screening test, and mislead his prospective employer as to his ability to hear. At other times workers will, for one of several possible reasons, respond in such a way as to exaggerate the extent and degree of his hearing loss. Either case can be detected and handled on an individual basis.

In addition to the above, the otologist participates in the medicolegal aspects of hearing claims as they arise. Some of the many thorny questions upon which he has been asked to render opinions are discussed on the following pages:

At what level of sound intensity, and over what period of exposure, does hearing damage take place? Until the subcommittees of the American Acoustical Society, and the American Academy of Ophthalmology and Otolaryngology, publish their final recommendations, only tentative figures are available for guidance. In general, it can be stated that the best damage risk estimates tend to be made in terms of the frequency spectra of factory noises, as well as the intensity. They show that decibel for decibel, high frequency sounds are more damaging and that sounds that contain many frequency components are less damaging than sounds which liberate the same energy within a narrow band of frequencies.

What was the status of the claiming employee's hearing when he came to work for the employer? This question was of crucial importance when the first wave of hearing loss claims appeared. At present there is no valid reason why employers should not require routine audiometric examinations as part of the pre-employment physical examinations. When cases arise, employers should, as a matter of defense, be able to establish degree and extent of any hearing loss present, when the worker was hired, the hearing status at the time of claiming, and the cause of any decrement in hearing between these times. The need of otological advice in such cases should be evident to all concerned.

What tests should be used for assessing the hearing loss? It is pretty generally agreed that older methods such as watch ticks, coin clicks, and whispered and conversational voice are too subjective to be of any value. Calibrated pure tone audiometry is much to be preferred to any of the above, but it is sometimes objected to, on the grounds that it measures acuity for tones rather than for useful everyday speech. Calibrated speech audiometry in the hands of skilled practitioners, is the method of choice, and is rapidly gaining endorsements from authoritative professional groups.

What formula should be used to evaluate the hearing test results in terms of percentage of hearing disability? The American Medical Association formula of 1947 has, in the past, been used by many otologists for evaluating pure tone audiograms; however, valid objections to the use of this formula have arisen, especially in connection with hearing loss claims based upon decreased acuity for high frequency tones. It is argued that such claims can be liberally awarded when this formula is used, although the claimant has lost little of his ability to follow normal conversation. In New York State, the Fletcher Point Eight (.8) method has been used. There is every indication, however, that for medico-legal purposes, speech audiometry will soon supplant pure tone audiometry with the sanction of national authoritative professional societies. It is expected, however, that pure tone audiometry will be used as an adjunct to calibrated speech audiometry.

Which of several hearing test results obtained by several doctors on different days should be used to evaluate the hearing loss? This question gains importance in cases involving claims for alleged occupational hearing loss. As the claimant is examined several times by different doctors in rooms of varying acoustical characteristics, and becomes familiar with the test procedures, variations in test results are commonly found. This gives rise to a diversity of opinions as to which of several different hearing curves should be accepted as most valid and reliable.

The Wisconsin Industrial Commission has ruled that when two or more differing audiometric results are found within a short period of time—performed under similar testing conditions—the test showing the best hearing acuity shall be used.

Are some workers more susceptible to noise induced hearing loss than others? That some workers are more susceptible to damage and loss than others can no longer be doubted.

How is the permanence of the hearing loss to be determined? The problem of permanency of hearing loss is difficult to solve. It is well-known that part of the total amount of measureable hearing loss that comes from noise exposure is recoverable. If the employee is removed from the noisy environment some improvement in hearing can be expected. The American Academy of Ophthalmology and Otolaryngology has expressed the following opinion regarding the question of permanency: "Hearing loss, caused by prolonged exposure to loud noise, may be considered as permanent six months following complete removal of the individual from the area of loud noise." A more definitive answer may be expected after an analysis of observations now being made by several researchers.

What consideration should be given to the age factor? Loss of hearing, which is an accompaniment of advancing age, has been given the name of presbycusis. This hearing loss in older people is supposedly due to simple atrophy and degeneration of the end organ. The hearing loss is of the perceptive type, affecting the higher frequencies and rarely involving the lower scale. This high tone loss has been demonstrated to increase with each decade of life, and appropriate allowances can be made for the amount of hearing loss which can be expected, due to normal aging of the hearing mechanism. An allowance of 5 per cent is made for individuals over 50 years of age and 10 per cent for the 60 and over age group.

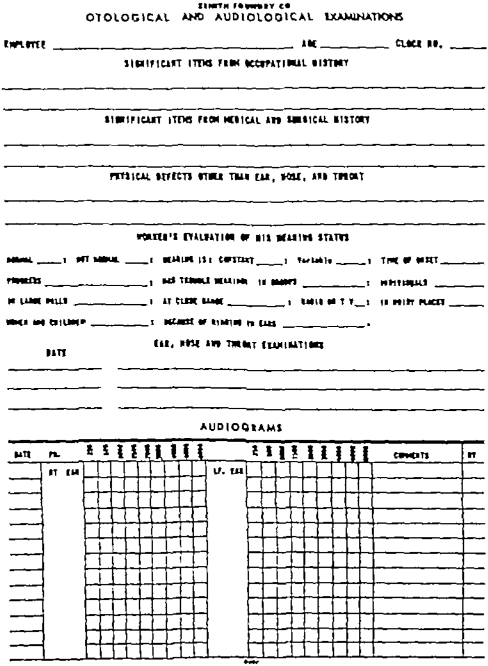
The hearing conservation program begins with a thorough study of the noise levels found in the various working areas. A preliminary survey reveals those areas where sound levels reach intensities that are potentially damaging. Then a more detailed analysis of these areas is made to determine the intensity, duration and frequency characteristics of the overall sound pattern.

Such an analysis indicates the amounts of energy present in the more traumatizing frequency bands, and determines the amount and type of acoustical treatment and individual worker-worn ear protective devices. Where noises are predominantly of the impact type, it is often necessary to use the cathode ray oscilloscope to obtain an accurate measurement.

The next step is to obtain a convenient and accessible room suitable for the performance of hearing tests. When a sufficiently quiet room cannot be found, it is sometimes possible to treat an existing space with acoustical materials and obtain an acceptable room for practical hearing tests.

The existing medical records are studied, and additional forms are prepared, which will allow the serial recording of periodic otological and hearing tests, and pertinent items of medical and industrial history for each worker (see Figs. 1

through 3). Experience has shown that charts such as these which allow the serial recording of initial test results, together with the findings of a dozen or more "follow-up" audiograms on a single sheet, are less bulky and less subject to loss than a group of single index cards for each test on the same individual. Since the results of all hearing tests given a worker are grouped together on a single sheet, any changes in hearing status are quickly and easily noted.

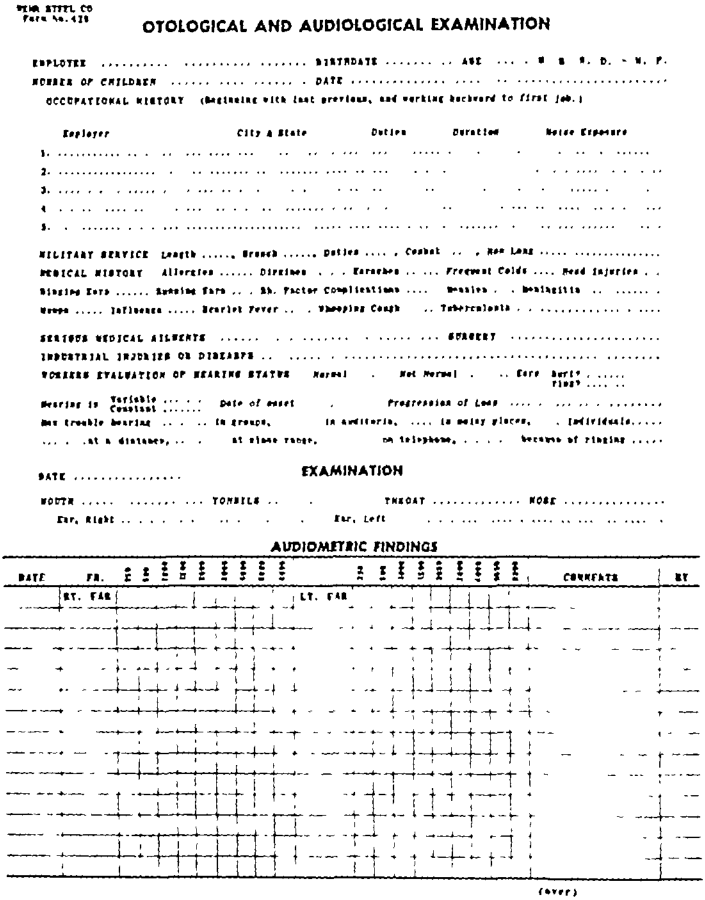


OTOLOGICAL AND AUDIOLOGICAL EXAMINATIONS

AUDIOGRAMS

Fig. 1

After a short and intensive course of instruction from the otologist, followed by supervised practice, plant personnel are ready to perform audiometric tests on workers. Close supervision is required at first, and is gradually relaxed as the tester develops skill and judgment.



[illegible]
Form No. [illegible]

OTOLOGICAL AND AUDIOLOGICAL EXAMINATION

EMPLOYEE ...... BIRTHDATE ...... AGE ...... M S W D ...... M. F.
NUMBER OF CHILDREN ...... DATE ......
OCCUPATIONAL HISTORY (Beginning with last previous, and working backward to first job.)

Employer	City & State	Duties	Duration	Noise Exposure
1.				
2.				
3.				
4.				
5.				

MILITARY SERVICE Length ......, Branch ......, Duties ......, Combat ......, How Long ......
MEDICAL HISTORY Allergies ...... Dizziness ...... Earaches ...... Frequent Colds ...... Head Injuries ......
Ringing Ears ...... Running Ears ...... Rh. Factor Complications ...... Measles ...... Meningitis ......
Mumps ...... Influenza ...... Scarlet Fever ...... Whooping Cough ...... Tuberculosis ......

SERIOUS MEDICAL AILMENTS ...... SURGERY ......
INDUSTRIAL INJURIES OR DISEASES ......
WORKERS EVALUATION OF HEARING STATUS Normal ...... Not Normal ...... Ears hurt? ......
Tire? ......

Hearing is Variable ...... Constant ...... Date of onset ...... Progression of Loss ......
Has trouble hearing ...... in groups, ... in auditoria, ... in noisy places, ... individuals, ...
...... at a distance, ... at close range, ... on telephone, ... because of ringing ......

EXAMINATION

DATE ......
MOUTH ...... TONSILS ...... THROAT ...... NOSE ......
Ear, Right ...... Ear, Left ......

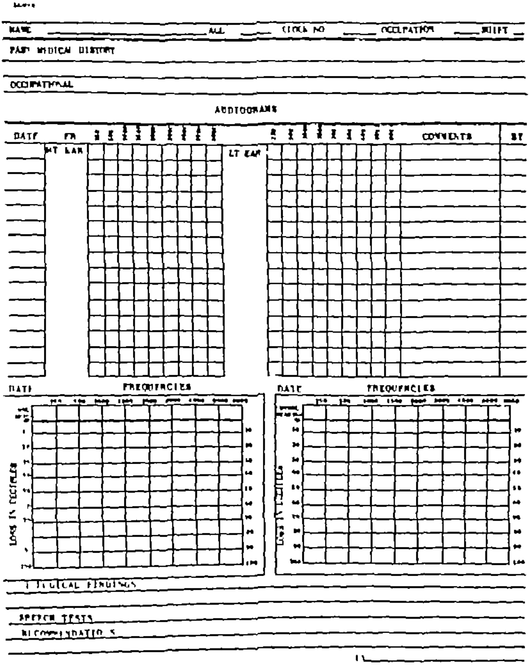
AUDIOMETRIC FINDINGS

DATE | FR. | RT. EAR | LT. EAR | COMMENTS | BY

(over)

FIG. 2.

The results of all hearing tests are reviewed and evaluated by the otologist, and all workers showing significant deviations from normal hearing are listed. When enough workers with abnormal hearing are listed to justify it, the otologist arranges to spend part of a day examining them in the plant. It has been found that this practice, in other words, the otologist's visiting the plant, is more economical and convenient than sending the workers to his office. In typical small plants the otologist need hold examinations only two or three times a year.



NAME ______ AGE ______ CLOCK NO ______ OCCUPATION ______ SHIFT ______

PAST MEDICAL HISTORY

OCCUPATIONAL

AUDIOGRAMS

DATE

RT EAR

LT EAR

COMMENTS

BY

DATE FREQUENCIES

LOSS IN DECIBELS

DATE FREQUENCIES

LOSS IN DECIBELS

OTOLOGICAL FINDINGS

SPEECH TESTS

RECOMMENDATIONS

FIG. 3.

The value of his supervision and guidance is amply demonstrated by the type of errors which he routinely detects. He must determine whether hearing loss is due to acoustic trauma or to normal aging of the ear, and whether a worker's hearing loss is real or only apparent. He is familiar with the effects of ambient noise, active respiratory diseases, obstructions to the ear canals, etc.

He quickly determines whether the worker is consciously or unconsciously exaggerating, either his hearing ability or his hearing loss, and he is alert to detect "cross-hearing," which can lead the inexperienced tester to credit a totally deaf ear with serviceable hearing; but in the final analysis, the worth of the aurist's contribution to the hearing conservation program can be judged by the extent to which he helps to achieve the three major objectives of a second hearing conservation program:

1. to conserve the hearing of workers,

2. to avoid unnecessary economic loss to the employer,

3. to collect the facts and data which will enlarge our knowledge of the defects of industrial noise, and how they can be overcome.

At all times, the otologist should be aware, as much as are his co-workers, that this is a joint effort requiring the earnest cooperation of workers and management and of the medical and allied professions. It will be his responsibility to interpret the data in hearing records made as part of the program and to share his findings with his colleagues. At the present time, the results of hearing tests on thousands of workers have been tabulated in the Wisconsin area alone. Together with similar data collected in other states, these statistical studies are beginning to fill in the gaps in our knowledge and to make speculation about the facts of hearing loss in industry unnecessary. It is to be hoped that more otologists will interest themselves and become active in attempts to overcome the effects of industrial noise. On the basis of my own experience, I would definitely recommend those interested to take part in our efforts. There are both challenges and opportunities aplenty in this field, and time alone will tell whether we as otologists have been equal to them.

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THE ROLES OF SENSATION LEVEL AND OF SOUND PRESSURE IN PRODUCING REVERSIBLE AUDITORY FATIGUE.

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The determination of unusual susceptibility to auditory fatigue has long been proposed as a prophylactic step in a noise control program. One of the first workers to standardize a test for individual differences in susceptibility was Smith,[3] who studied threshold shifts at 4000 cps. caused by stimulation at 2000 cps. for two minutes, finding fatigue to range from 10 to 56 db. This wide range opened the possibility of an industrial program designed to reject those who suffer the greatest fatigue. Peyser[2] determined threshold at 256 cps., stimulated at 80 db intensity for five minutes, and 30 seconds later again determined threshold. Of 80 subjects, seven showed threshold shifts of more than 5 db.

Wilson[4,5,6] has energetically attacked this problem with rather large numbers of subjects. He early discarded testing at 256 cps., finally standardizing a stimulus of 2048 cps. at 80 db intensity for five and eight minutes. Threshold shifts at 4096 cps. were determined as an indication of fatigue.

By comparing irreversible audiometric loss caused on the rifle range with amount of fatigue on his tests, Wilson claimed to have established a good predictive relationship between the two functions. If true, Wilson's test, or a similar one, could well serve in industry and in the armed forces to reduce the number of auditory casualties and compensable cases.

A question arises in a test of this sort whether it is well to place the stimulating frequency at a certain intensity for all subjects, or to place it a certain number of decibels above the individual's own threshold. It is clear that if a subject has a threshold 10 db better than "normal," and another subject has a threshold of 10 db less than "normal," the same

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physical intensity of, say, 80 db on the audiometer scale will furnish sensation levels of 90 and 70 to the two subjects respectively; now, if the fatiguing effect of a tone depends not on its physical intensity but on its perceived *loudness*, then the effective stimulus conditions for the two subjects will have been greatly different, and we shall find we have not given comparable fatiguing conditions as we wish to do. This study proposed to investigate the relative effectiveness of loudness and intensity in producing auditory fatigue. This done, the data should help to standardize a fatigue test to attack the problem of predicting irreversible trauma.

PROCEDURE AND APPARATUS.

Thresholds on a total of 66 subjects were obtained at 2048 and 4096 cps. with a Sonotone audiometer calibrated in this Laboratory. The range of hearing loss at 2048 cps. was — 10 to + 65 db. A fatiguing stimulus at 2048 cps. was led to each subject for five minutes from a Hewlett-Packard oscillator connected to the Sonotone earphone. Sound pressure level (as measured in the National Bureau of Standards 9A Coupler) was maintained at 97 db (this is the equivalent of 80 db above "normal" on American audiometers at this frequency). The stimulus was terminated by a two-way switch that immediately fed a 4096 cps. tone to the earphone from the Sonotone audiometer. The level of this tone had previously been set 20 db above the individual subject's threshold for that frequency. The 4096 cps. tone was turned on and off in ¼-second intervals; the pulsing nature of this tone made it easier to distinguish in the presence of high-tone tinnitus, which very often follows stimulation of the sort used here. As soon as the 20 db sensation level tone was heard, the subject tapped a microphone of a wire recorder, whereupon the experimenter reduced the tone 5 db and waited for another tap; and so on until the time taken to return to within 20, 15, 10, 5, 2.5, and 0 db at threshold had been recorded on the wire. In this way, a complete picture of every man's recovery curve could subsequently be drawn.

If a subject heard all tones as soon as presented, we assumed that his recovery curve was complete in less than 10 seconds, the time it required to move the audiometer dial from

20 to 0 db in the steps as given above. In that case, one or more repetitions of the whole procedure were given, in which the dial was set initially at 10 rather than at 20 db.

RESULTS AND DISCUSSION.

Effect of Sound Pressure Level Versus Sensation Level.

Since all of our subjects received the same acoustic stimulus, distributions of various indexes of fatigue will serve to specify the effect of sound pressure level. Table I gives the distributions of times to return to own threshold, and to within 5 and 10 db of own threshold. From these data one can fairly well infer the effect of sound pressure level *per se* on the recovery curve. Note that about 58 per cent of our first 48 subjects completely recovered in less than two minutes. If, however, we consider only those 25 men whose hearing at 2048 was *better* than "normal," we find that only 40 per cent of this exceptionally acute group recovered in less than two minutes. Evidently the data of Table I are strongly influenced by selection of subjects, and we conclude that sound pressure level is no sure predictor of fatigue effects.

TABLE I.

INDIVIDUAL DIFFERENCES IN RECOVERY TIME AT 4096 CPS. TO STIMULATION WITH 2048 CPS. AT 97 DB SOUND PRESSURE LEVEL FOR 5 MINUTES

TIME TO RECOVER TO.					
1 Own Threshold		2 Within 5 db		1. Within 10 db	
Time	N	Time	N	Time	N
0-2′ (1′59.9″)	28	0-30″ (29.9″)	29	0-20″ (19.9″)	30
2′ - 4′	7	80″ - 60″	6	20″ - 40″	4
4′ - 6′	4	60″ - 90″	2	40″ - 60″	0
6′ - 8′	4	90″ - 120″	2	60″ - 80″	1
8′ - 10′	2	120″ - 150″	2	80″ - 100″	2
10′ - 12′	1	150″ - 180″	1	100″ - 120″	1
12′ - 14′	0	180″ - 210″	2	120″ - 140″	0
14′ - 16′	0	210″ - 240″	2	140″ - 160″	0
16′ - 18′	2	240″ - 270″	2	160″ - 180″	0
				180″ - 200″	1

When we consider the relative effect of sensation level, it is necessary only to group our subjects according to individual threshold at 2048 cps. It will be recalled that the threshold range of our subjects at this frequency was minus 10 to plus 65 db ; therefore, the sensation level of the 80 db tone on the audiometer actually ranged from 90 db to 15 db SL. One would assume that the subject who is presented with a 15 db tone will show little fatigue in comparison with the subject who hears a 90 db tone. Fig. 1 shows mean curves for 36 of our subjects divided into five groups according to their pre-stimulation threshold (the remaining 12 of the 48 subjects who took this test had hearing losses at 2048 cps. which did not fit into the categories of Fig. 1).

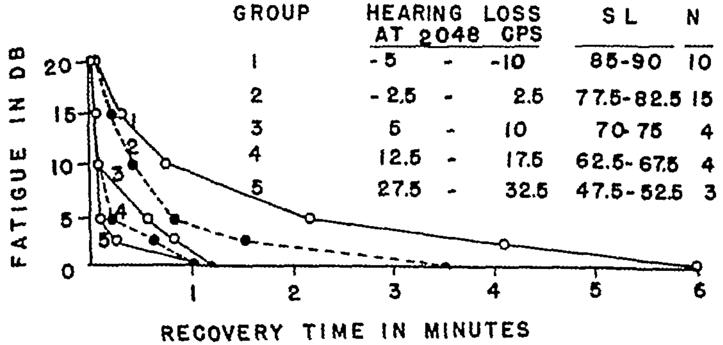

Fig. 1. Effect of Sensation Level on Recovery.

One can see the very significant differences among at least the first three groups with highest sensation level. Complete recovery time for the highest sensation level group (85-90 SL) is 360 seconds; for the next highest group it is 212 seconds and for the third highest group it is 80 seconds. The other three groups whose hearing is within 10 decibels of normal at this frequency, all show such rapid and similar recovery curves that one can say, when the tone is about 60 db sensation level and below that there is little difference in recovery from this stimulation, as measured by our technique, and that there is no significant amount of fatigue. At 60 db SL and less, the difference occasioned by sensation level can not be assessed by this technique, but above this level sensation level must be taken into consideration before predicting any sort of susceptibility to fatigue based on a stimulation set at 80 db above "normal" on the audiometer.

Individual Differences.

The usual wide range of individual differences appears in our data. The raw data for six representative subjects in Group 2 of Fig. 1 (those who were "normal" ± 2.5 db) are shown in Fig. 2. The recovery time to own threshold ranges from seven seconds to 16 minutes. Note that this wide variability occurs even among subjects who were given substantially equal stimulation, whether measured by sound pressure level or by sensation level.

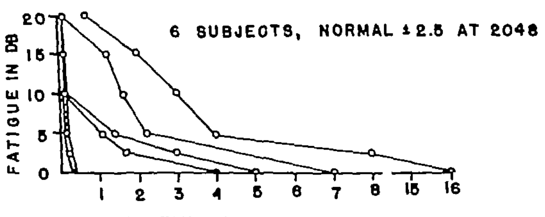

Fig. 2. Individual Differences in Susceptibility to Fatigue.

The effect of type of deafness was not investigated in this study, though we did include a few partially-deaf individuals to show with especial clarity the difference between equal sensation level and equal physical intensity. The problem of type of deafness has, however, been well covered by Huizing,[1] who gave the same sensation level of stimulation to patients with about the same hearing loss (50-60 db) but with quite different types of disorder. Pure conduction deafness yielded 7-12 db fatigue (about the same as normal-hearing individuals on the identical test), and inner ear deafness without recruitment yielded 5-11 db, while inner ear deafness *with* recruitment yielded 19-22 db. Huizing concludes that the fatiguing effect of a stimulus depends among other things upon its *loudness.*

Comparison of Wilson's and the Present Study.

Wilson used subjects with no hearing complaints and his threshold loss was no more than 30 db at 4096 cps. in either ear. His experimental procedure was to determine the subject's threshold at 4096 cps. in both ears, then to expose the ears (one at a time) to 2048 cps. for five minutes at "80 db intensity." Threshold was "immediately" taken at 4096 cps., and again ½, 1, 2, 3, 5, and 8 minutes following the start of recovery, or until 10 db from threshold was reached. (A deviation of less than 10 db from the pre-stimulation threshold was considered to be within experimental error.) This procedure was then repeated on the opposite ear.

Wilson arbitrarily divided his subjects into three groups depending on the time required to return to within less than 10 db of threshold (we presume this to be subject's threshold, not necessarily audiometer "normal").

When, regardless of threshold at 2048 cps., we group our data in the same fashion as Wilson, comparable results appear as in Table II. This table reveals disagreement between the two studies. Only one of our cases did *not* meet Wilson's criterion of no fatigability (i.e., return to within less than 10 db of own threshold in less than two minutes), whereas, 30.8 per cent of Wilson's cases did not meet this criterion (sum of Groups 2 and 3).

TABLE II.
COMPARISON OF WILSON'S AND THE PRESENT STUDY.

Group	Percentage of Cases Wilson	Present Study	Returned to within less than 10 db of Threshold in:
1	69.2%	97.9%	Less than 2 minutes
2	14.8	0	2 - 3 minutes
3	16.0	2.1	3 or more minutes

These discrepancies can hardly be due to differences in the 2048 cps. threshold between the two experiments. It might be thought for example, that most of Wilson's subjects were better than "normal" on the audiometer, and then in consequence if 80 db over "normal" were used, the actual sensation level would be well over 80 db; however, we used ten subjects who were better than "normal" by at least 5 db, and who consequently received stimulation at 85 db or more sensation level. None of these required more than two minutes to reach 10 db over their own threshold. We may further consider our 15 subjects who were "normal" ± 2.5 db, and so received a stimulus at 80 db sensation level (within experimental error), and compare this group with Wilson's on the assumption that what he did was to stimulate at 80 db above each subject's threshold. Only one of this group (7 per cent) did not meet Wilson's criterion, again as compared with his 30.8 per cent. The differences then are not those concerned with variations in sensation level or sound pressure level of stimulation.

Wilson's percentages may be expected to be somewhat larger than ours, since he included a man in the group dictated by the more susceptible ear if there was a difference in the ears. We used only one ear from each subject.

The writer feels, however, that most of the difference in the studies is probably due to the tinnitus which easily can partly obscure a continuous tone. It will be recalled that ours was a pulsing tone which serves to avoid penalizing those not-rare individuals with tinnitus at or near 4096 cps. after loud stimulation. Wilson mentions a number of subjects, in whom tinnitus suddenly ceased after 2-3 minutes, whereupon their thresholds returned sharply to normal.

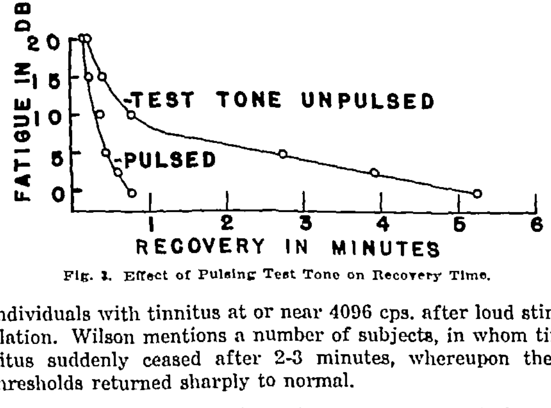

Fig. 1. Effect of Pulsing Test Tone on Recovery Time.

In order to investigate this point, we used an entirely new group of 18 subjects, and gave them stimulation at 2048 cps. at 80 db sensation level for five minutes, immediately following with a 4096 cps. test tone, either pulsed as described previously, or continuous. A direct comparison in this group of 36 ears can then be made in the recovery time as investigated by a pulsed versus a non-pulsed tone. The data is summarized in Figure 3, and show that the median recovery time is of the order of 45 seconds for a pulsed tone, but is of the order of 315 seconds for a continuous tone. The differences between the two techniques are somewhat greater if the arithmetic mean is taken, rather than the median. This for the reason that several of the subjects had a tinnitus at the 4000 cycle region, which interfered for many minutes with hearing the unpulsed tone at all. These extreme subjects, however, do not unduly influence the data in the median measure of central tendency.

Another way to consider the data of these 18 subjects is to compute the percentage of those who returned to normal, according to Wilson's criterion. With the pulsed technique, all of the 36 ears had returned to within less than 10 db of threshold in less than two minutes. On the other hand, with the unpulsed technique only 22, or about 61 per cent, returned to this criterion. The other 39 per cent required longer time. It may be observed that this percentage is approximately what Wilson found, as revealed in Table II.

Criterion of Fatigability.

In choosing a criterion of fatigability for the practical purpose of screening individuals particularly susceptible to fatigue, Wilson settled on the threshold shift of 10 db or more, after two minutes of recovery. We feel that a somewhat more stringent criterion would perhaps be preferable. As the raw data show, a one-minute interval would have picked out five ears, or about 10 per cent, as being especially susceptible. This percentage is in general more usable in a personnel selection program than the 2 per cent picked out at the two-minute interval (see Table II).

An alternative is to record the time to return to within 5 db. of own threshold. Table I shows that only about four to five minutes would be required for even the most susceptible. Choosing this level would not, then, prolong the test unduly; and the distribution of recovery time is wide enough to serve in a statistically controlled fatigability-research program.

We conclude that either criterion could serve, the threshold after one minute, or the time to return within five db of threshold, depending upon the convenience of available apparatus.

SUMMARY AND CONCLUSIONS.

A test for screening fatigue susceptible individuals was investigated. A tone of 2048 cps. was sounded for five minutes at 97 db sound pressure level (80 db on the audiometer). The individual's recovery curve was carefully plotted at 4096 cps., the region most affected by such stimulation. Results were analyzed in terms of sensation level (data of their own threshold), and physical intensity. Sensation level proved the more important parameter.

Considerably more rapid recovery was found than reported previously by Wilson, who used, as a criterion of fatigue, inability to hear a tone 10 db over own threshold after two minutes recovery. The present study shows a better criterion to be, either the time in seconds to return to within 5 db of own threshold, or the residual hearing loss after one minute.

It is of considerable importance to use a pulsing tone rather than a steady tone in determining threshold during recovery from stimulation, in order to minimize the irrelevant masking effects of the high tone tinnitus which very often follows stimulations such as used here.

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CHEMICAL BURN OF THE ESOPHAGUS. REPORT OF A CASE TREATED WITH CORTISONE AND PENICILLIN.*

A. F. JUDGE, M.D.,

Martinsburg, W. Va.

The majority of chemical burns of the esophagus fall into the class of second-degree burns. Stumboff,[1] in a series of 603 cases, found that burns of the second degree constituted 63 per cent of the total.

In a second-degree burn, necrosis of the mucous membrane and muscular layer takes place. There is a sloughing of the necrotic tissue followed by the formation of granulations. Collagen fibers appear and form the basis for scar tissue formation. These fibers appear usually about the second week following the burn. Unless the formation of scar tissue can be prevented or controlled, a stricture of the esophagus results.

Stothers,[2] in 1952, reviewed the prophylaxis of stricture formation following burns of the esophagus, and stated that it was generally conceded that early bouginage by the Salzer method is the best means available to prevent stricture following chemical burns. Dilation by any means usually takes months and possibly years.

Krey[3] by animal experiments, showed the importance of secondary infection in the formation of cicatricial strictures. Inflammatory changes produce granulation tissue, which in turn is transformed into scar tissue. Limitation of inflammatory reaction by resting the burned esophagus until epithelization is complete, retards the formation of scar tissue. In a group of animals having a burn of the esophagus and treated by gastrostomy, sulfonamides, penicillin and streptomycin, epithelization took place in 35 days, and only moderate stenosis was observed. Stenosis of the esophagus sufficient to

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* From the Department of Otolaryngology, Veterans Administration Center.

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prevent feeding developed in three other control groups. The first group received no treatment; the second group received antibiotics; the third group received antibiotics and an indwelling tube.

Rosenberg, Kunderman, Vroman and Moolten[4] administered large doses of cortisone to rabbits with chemical burns of the esophagus and found that fibrous stricture formation was remarkably diminished; however, many of the animals died as a result of infection. Infections were rare in the control group.

In a later series of experiments, Rosenberg and associates[5] found that the administration of penicillin with cortisone to rabbits having chemical burns of the esophagus prevented stricture formation, and also improved the mortality figures. In a small number of rabbits receiving penicillin and cortisone, spontaneous rupture of the esophagus occurred in the second or third week, usually resulting in mediastinitis and death.

Weisskopf[6] treated a child with a severe lye burn of the esophagus. He employed cortisone, terramycin and a Levine tube. The esophagus healed without stricture formation or other complication. He then produced lye burns of the esophagus in dogs. The treated animals received cortisone and terramycin. The controls received no medication. Animals in the control group showed marked granulations of the esophagus in the early stage, and developed strictures if allowed to live over five weeks. No stricture developed in the treated animals.

Recently we had under our care a patient with a severe second-degree burn of the esophagus due to sulfuric acid. The results of treatment were so satisfactory as to merit a report.

REPORT OF A CASE.

R. D., white, male, age 25, was admitted to the Neuropsychiatric Service on July 14, 1953, because of a depressive state, in which he had attempted suicide by drinking sulfuric acid used for storage batteries. This episode had occurred on the day prior to admission. He had received emergency treatment at a local hospital and was then transferred to the V. A. Hospital. Upon admission temperature was 100.6°, pulse

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80, and respirations 20. The next day the temperature was elevated to 101°. Blood pressure was 126/80. He complained of sore throat and inability to swallow. Liquids when taken by mouth were vomited almost immediately.

Because of the dysphagia, he was seen in the E. N. T. Clinic on July 15. Examination of the pharynx showed the mucosa to be markedly congested. In the hypopharynx the mucosa was edematous as well, and there was profuse saliva stagnant in the valleculae. The patient was started on procaine penicillin 300,000 units daily by intramuscular injection, and cortisone 50 mg. twice daily. He was given bland liquids by mouth. Sore throat and dysphagia were less marked in 48 hours.

On July 20, 1953, esophagoscopy was performed. This was seven days after the acid had been ingested. The mucosa of the entire esophagus was congested and edematous. At a level of 27 cms. from the upper teeth there was marked sloughing of the mucosa. Near the hiatus the mucosa was again found to be sloughing in the entire circumference of its lumen. The sloughing areas bled on passage of the esophagoscope.

A polyethylene Levine-type feeding tube was passed through the esophagoscope into the stomach and the esophagoscope withdrawn. The feeding tube was then brought out through the left nostril by means of a catheter. The patient was fed a liquid diet high in protein and meeting the caloric, mineral, vitamin and fluid requirements. The cortisone and penicillin were continued daily. There were no physical complaints. On August 6, the feeding tube was removed and the cortisone and penicillin discontinued. By this time the patient had gained 15 pounds in weight.

Esophagoscopy on August 18, showed a normal mucosa with no evidence of scarring, nor of stricture. This was five weeks after the patient had swallowed the acid.

The patient was discharged from the hospital on August 19, 1953, with instructions to return for periodic follow-up; however, all efforts to induce him to return for esophagoscopy and esophagram were unavailing. He was interviewed at his home in December, 1953, by a social service worker. At that time he stated he was having no difficulty with his regular diet, and that his weight had remained constant. This was five months after the trauma to the esophagus.

COMMENT.

Acid burns of the esophagus are less frequently the cause of stricture of the esophagus than are lye burns; however, from the appearance of the esophagus in this case, we feel that in the ordinary course of events, stricture would have resulted, or months and possibly years of dilation would have been required to prevent it.

Polyethylene tubing is chemically inert and causes no tissue reaction. It maintains the patency of the lumen, puts the ulcerated mucosa at rest and allows the patient to take his normal fluid and caloric requirements without discomfort. Broad spectrum antibiotics may be given with the tube feedings. Its use probably also lessens the chance of perforation.

Weisskopf[6] used terramycin in his case and also in his experimental animals to control secondary infection, we used penicillin Apparently, either broad or narrow spectrum antibiotics are efficacious in controlling secondary infection and resulting granulations Used alone, they probably will not effectively prevent stricture formation

Cortisone lessens the formation of collagen fibers and resulting scarring, and when used alone, in animal experiments, resulted in high mortality In our case it was administered along with the antibiotic from the third day after the burn until the twenty-fourth day The dosage used was small, because we did not wish to submit the patient to the numerous and daily laboratory tests required by hospital regulations when a full dosage of cortisone or ACTH is administered

SUMMARY.

A case of second-degree burn of the esophagus, which was treated by cortisone, penicillin and polyethylene feeding tube, and which healed without stricture formation is reported

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SUBACUTE MASTOIDITIS.

GEORGE S. RICHARDSON, M.D.,
Albuquerque, N. M.

Ballance[1] quoted Celsus as having said, "Though we should not refuse to give modern authors due credit for their discoveries or happy imitations, it is none the less just to restore to the ancients what properly belongs to them."

This brief paper will deal with the clinical, audiometric and radiologic considerations for surgical intervention in the recent and recurrent mastoid infection. Clearer concepts on this subject are needed. The old indications for simple mastoidectomy in the pre-antibiotic era are obsolete. Every patient with a chronic ear wishes something had been done surgically during the simpler phases of the infection. There is no more shock to a simple mastoidectomy than to a tonsillo-adenoidectomy. Every purulent, acute otitis media is accompanied by mastoiditis, and the indications for surgery and the anatomy of the mastoid have become confused by the administration of antibiotics. The candidates for modified and radical procedures are younger each year.

"Colds," sore throats and pink eardrums constitute many of the indications for penicillin and other antibiotic administration. The early treatment of these symptoms, by whatever specialty, is most important for the patient.

There is a general tendency to minimize the solid clinical judgment of the otolaryngologist in evaluating the need for surgery.

The final evaluation of the antibiotics and their promiscuous administration awaits the passage of time. The agreeable sophistry is advanced that the patient gets well faster with antibiotics, but has he acquired any natural immunity during their administration?

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BACTERIOLOGY.

In an address before the staff of one of our hospitals on this same subject in August, 1951, attention was called to the dangers of moniliasis from antibiotic administration. That danger undoubtedly still exists; if not a factor in otology, witness the rumbling of millions of feet of intestine.

An editorial[2] in a reputable medical publication, stated: "The replacement of respiratory or gastro-intestinal flora with antibiotic resistant strains of pathologic organisms such as hemolytic *staph. aureus* and hemolytic streptococci is potentially a much more serious problem than overgrowth of normally saprophytic fungi or yeastlike organisms."

Not only are routine ear cultures bringing these strains to dock but there are increasing numbers of *proteus mirabilis, proteus vulgaris, ps. aeruginosa, alcaligenes faecalis, B subtilis e. Coli* and *aerobacter aerogenes* infections of the middle ear and mastoid.

Ellard Yow[3] called attention to the increased importance of *Proteus* and *Pseudomonas* as pathologic organisms, isolating these from normally sterile body fluids during antibiotic therapy. He felt that surgical drainage and removal of non-vital tissue were important in controlling these infections.

An increasing number of well-known authors have been pleading for surgical treatment of surgical lesions, *i.e.* pus-filled cavities, but their cries go unheeded.

A case was recently seen of purulent mastoiditis, of ten years' duration, with an intact drum except for a sieve-like group of perforations high posteriorly, which revealed *proteus mirabilis.* The patient had been given everything including cortisone. The aural discharge may still persist. Another patient in whom a bilateral secondary mastoidectomy revealed a middle ear and antrum on both sides—behind intact drums—filled with the thick and typically tenacious secretion suggestive of *ps. aeruginosa*, was reported "no growth." The bacteriology laboratory may have poor laboratory technique

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An editorial[2] in a reputable medical publication, stated: "The replacement of respiratory or gastro-intestinal flora with antibiotic resistant strains of pathologic organisms such as hemolytic *staph. aureus* and hemolytic streptococci is potentially a much more serious problem than overgrowth of normally saprophytic fungi or yeastlike organisms."

Not only are routine ear cultures bringing these strains to dock but there are increasing numbers of *proteus mirabilis, proteus vulgaris, ps. aeruginosa, alcaligenes faecalis, B subtilis e. Coli* and *aerobacter aerogenes* infections of the middle ear and mastoid.

Ellard Yow[3] called attention to the increased importance of *Proteus* and *Pseudomonas* as pathologic organisms, isolating these from normally sterile body fluids during antibiotic therapy. He felt that surgical drainage and removal of non-vital tissue were important in controlling these infections.

An increasing number of well-known authors have been pleading for surgical treatment of surgical lesions, *i.e.* pus-filled cavities, but their cries go unheeded.

A case was recently seen of purulent mastoiditis, of ten years' duration, with an intact drum except for a sieve-like group of perforations high posteriorly, which revealed *proteus mirabilis.* The patient had been given everything including cortisone. The aural discharge may still persist. Another patient in whom a bilateral secondary mastoidectomy revealed a middle ear and antrum on both sides—behind intact drums—filled with the thick and typically tenacious secretion suggestive of *ps. aeruginosa,* was reported "no growth." The bacteriology laboratory may have poor laboratory technique or, more significantly, the cultural characteristics of bacteria are likewise changing.

CLINICAL PICTURE.

The case seen by the otolaryngologist usually has a history of recent antibiotic administration.

Chief among the presenting symptoms of this type of infection is *pain*. It may be located in the classical areas of old —tip, emissary vein, and/or root of zygoma. On specular examination it may be seen surrounding the bony canal in the absence of external ear infection, and manual pressure may elicit a wince or exclamation.

The *psychodynamic* factor must be excluded, as this idea clouds the symptom as frequently as do the antibiotics. Whether inflation relieves the pain is a differential point.

The last "no growth" case mentioned above was believed to be of psychosomatic origin, although surgery (in spite of intact normal appearing drums), revealed cortical erosion of an unoperated side, bilateral, healed peri-sinus abscesses, thick discharge in aditus, middle ear and antrum, and intact trabeculae of bone. Operation resulted in complete relief of all symptoms, notably night pain.

Night pain often awakens the patient from sound sleep— as might a night terror symptom. It may be of a "thumping" character, if a sterile discharge is imprisoned in the tip from escape to aditus and is being replenished by secretion from seemingly "normal" mucoperiosteum of cell.

Temperature is no longer a prominent finding unless the chronic ear has progressed to jugular sepsis, brain abscess, subperiosteal abscess, or Bezold's type.

Aural discharge is not the reliable surgical indicator it once was. There is still a tendency to believe that this is part of a "tubal catarrh," from the perforation location. Many feel that every aural discharge is accompanied by mastoid disease. It is important to note its chronicity as it continually recurs, despite seemingly free intervals, usually in the same ear.

Page[4] feels that a continued but scant discharge through a minute "pouting" perforation is an indication for drainage of the tympanum through the mastoid to preserve the hearing and to prevent a chronic purulent otitis media. The white count may or may not be significantly elevated.

An objective finding of some worth is the appearance of the drum. There is frequently lysis of the structure with a slight ring of tissue; again, there may be a *wet tissue paper* appearance with evidence of moisture. It is sad to note such changes in patients before their fifth birthday.

Rutherford[5] has given a clear, concise and forthright picture of the management of acute otitis media.

The combined picture is one of much indecision on the part of the physician, a confused patient, and recurrences of trouble in the same ear over long periods of time. In one case still under observation in Pennsylvania (mostly by hearsay) this picture has persisted without surgical intervention for thirteen years. The greatest unaswered question is why there should be disagreement—not with patient or parent—but among physicians, as to the *need* for surgery.

AUDIOMETRIC.

The audiologic findings in these patients is nearly always constant if the drums are intact. In most patients, seen at bi-annual visits, who finally submitted to surgery and who are no longer bothered with ear pains or psychosomatics, certain audiometric findings are prominent.

In two ears in the same patient which seem equally affected, *i.e.* with retracted, lustreless, red or scarred drums and with same clinical findings, the audiometer will serve to reveal the more involved ear. Pullen's[6] observations are of value.

We know that a patient with an acute otitis media will have a decreased air conduction with possibly no change in bone conduction, but as the mastoid becomes involved, his categories of altered bone conduction are important. Pullen lists the large cellular mastoid which signifies extensive loss of bone conduction and the small, underdeveloped and sclerotic mastoids in which bone conduction is normal or only slightly impaired. In a chronic otitis media, "healing" through sclerosis, Pullen's observations will prevail; however, in the large, cellular mastoid filled with the "sterile" or "banked fire" of a mastoiditis, bone conduction will be better in the more extensively involved ear presumably because the insulating effect of the air cells is thus removed, and vibratory sound is transmitted more readily through the bone with added granulation or thick cell wall.

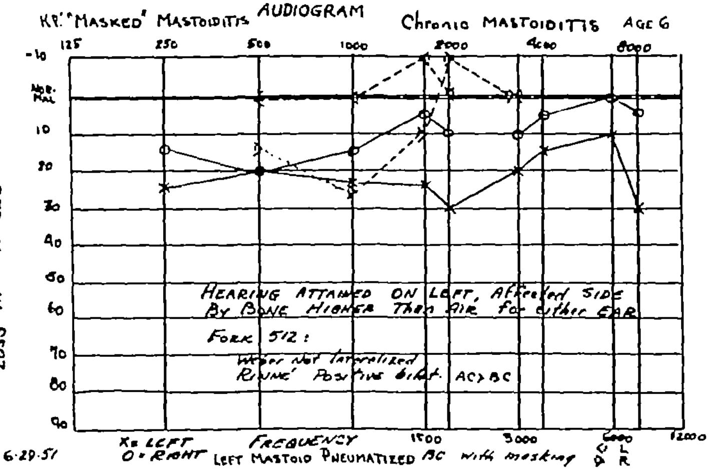

Fig. 1: K. P. Pre-operative audiogram on patient with diagnosis of left masked mastoiditis shows a very definite increase in Bone Conduction on left; diminished air conduction on left in absence of signs or symptoms. This increased bone conduction may be a characteristic audiometric feature of masked mastoiditis following long antibiotic therapy.

This observation has been confirmed at surgery in at least four patients and in many more when the radiologic comparison of mastoids was utilized to ascertain the ear of greater involvement on area of increased density basis.

Fig. 1 shows a typical observation in an operated patient. Hearing by bone conduction in the poorer ear was better than by air conduction in either ear.

RADIOLOGIC.

Boies and Younger[7] write of abnormal pneumatization of the temporal bone. It is their belief that in a population between the ages of five years and early adult life approximately

14 per cent probably have a middle ear mucosa in one or both ears that is not the thin mucoperiosteum of the well pneumatized mastoid. In a personal communication Boies states that he does not feel that lack of pneumatization, due to the effect of infection on a pneumatic structure, will necessarily carry a hereditary factor.

Two Spanish-American families have presented themselves, in which the mother and both sons have a discharging ear. No other members of these families have been seen with this complaint of aural discharge, yet here are two mothers and four sons, with three right ears and three left ears affected in a family pattern.

Boies feels that 14 per cent of such cases will show a deviation from normal. Since the postulation of Witmaack's theory there has been no unanimity of opinion on the significance of the sclerotic mastoid. Some prominent writers feel that it is an effort to heal a diseased area.

Tumarkin[8] quotes the theories of Diamant and Dahlberg regarding pneumatization and the pattern of the adult being stamped on the newborn; that acellular mastoids were simply normal variants genetically determined—a "sterilizing hypothesis" carried out with greatest care. Cawthorne, quoted in the same article, feels that chronic otitis media is the result and not the cause of an acellular mastoid.

Whatever the ultimate end, it is far better to dissociate theory from practical observation. The majority of patients will demonstrate a pneumatic mastoid by X-ray and, until proven otherwise, any deviation from this dawn-of-history-to-present pneumatic development should indicate mastoid pathology. Establishing time of onset of symptoms from history, physical findings, and the clinical picture, are still most reliable. X-ray studies are important for future evaluation.

Ersner, Alexander and Myers[9] decry the tendency to rely solely on X-ray in "irreversible mastoiditis" and Barrett[10] was among the first to point out the first reliable roentgenologic indicator in "masked" mastoiditis, of penicillin therapy.

In the young individual with an antrum at the two o'clock position there is an area of translucency surrounded by thickened sinus and dural plate. There is ordinarily an area of softening along the posterior canal wall with elevation and irritation of periosteum to produce clinical evidence of specular pain formerly accompanying "sagging of the canal."

For other than otologists this is frequently interpreted by the radiologist as "no evidence of cellular breakdown," when even acquainted with presence of an aural discharge in the radiographed patient. Law was the only radiologist who saw his temporal bone cases at surgery.

In the young adult with thick, hazy cellular partitions, some antral softening and increased density of bone surrounding the posterior canal, if the comparative film is normally thin in trabeculae with changes manifest equally in tip views, one may feel that this represents a pathologic condition. These are the insidious changes in the mastoid picture which, as stated by Ersner, are frequently overlooked. These are the mastoids which may terminate in mastoiditis or one of its intra-cranial complications.

Boies and Younger feel that abnormal pneumatization is an important factor in the chronicity of certain cases of sterile fluid in the middle ear; that it may play an important role in those children developing suppuration in the antibiotic era.

It is my feeling that the sterile fluid found in many of these mastoids at surgery is a pathologic sequence of antibiotic, sterile irritant fluid with bony change and progression to chronicity. How else may we account for the yellow color of the thin fluid entrapped in cells with apparently normal periosteum and filled with granulation? In most instances the radiologic haziness is revealed at operation as bleeding granulation, with thick, bony septae and pin-point areas of granulation. All of this is on the surface of the normally delineating boundaries of the mastoid cavity.

It must be remembered that even before antibiotics, the radiographic interpretation was not the sole criterion of operability and, like Tamari and Henner,[11] a uniform approach to the need for surgery is advocated before the ear becomes chronic and to indicate "when surgery is indicated."

THE SURGICAL POSITION.

The incidence of acute otitis media terminating in acute mastoid surgery (simple mastoidectomy) in the pre-antibiotic era was approximately 2 per cent. There have been decreased numbers of mastoidectomies at institutions like the Manhattan Eye, Ear and Throat Hospital since 1947. There is a need for better surgical indications in the outlying areas where reliance is still being placed on the antibiotic alone, without preliminary myringotomy, when needed.

A tabulation of mastoidectomies done by this writer in the five-year period, August 8, 1948 to 1953, is listed for 5,500 new patient office registrations.

One case was done in 1948, three in 1949, ten in 1950, 19 in 1951, 12 in 1952, and 3 thus far in 1953. Three of these were radical mastoidectomies; the remaining cases were simple mastoidectomies. The incidence of those coming to surgery is less than 1 per cent and these were by no means, all acute cases. Only six were subperiosteal abscesses and one was a brain abscess. The downward trend for 1952-53 reflects an attitude of indifference and inability to "reform" those who rely on antibiotics.

Probably as many refused surgery as had the operation, and the incidence of those who needed surgery has apparently been reduced but very little from the pre-antibiotic era. Those refusing surgery rarely return for evaluation, and it is this group which surgery would benefit in arresting the progress of pathology. Those having surgery usually have tried everything else, and surgery comes at the stage of urgency—a rare thing in antibiotic days, or when all other courses of treatment have been exhausted. In most cases the hearing is down; drums are nearly gone and only complications are prevented.

There have been innumerable authors urging that a pus-filled lesion is a surgical lesion, and nonetheless true for the mastoid infection. It is comparable to osteomyelitis only in the fact of bony involvement, and is worse from a standpoint of the tremendous vascularity of the bone, surrounding vital structures and the tragic loss of hearing in the young from damage to the delicate mechanism.

There may be only a remnant of drum left before mastoidectomy is suggested by the general physician, and the case is referred to the otolaryngologist. Simple precedures will no longer produce drum healing.

We have urged operation on all patients with subperiosteal abscesses, as well as those exhibiting marked bony change, without treatment by the antibiotics for a month or more, in which the conservative measures have failed to produce a dry ear.

We have had no serious complications save brain abscess to convince the patient as to the wisdom of surgery. We have also felt that in the patient with recurrent attacks of otitis media, in the same ear, surgery is the wiser course. When these attacks number two or more in a year or less, and despite previous conservative or antibiotic therapy, surgery is indicated. In one of my cases which has been seen at yearly intervals the pediatrician has averaged a course of penicillin monthly for each "pink" eardrum. There appears to be an increase of adhesive processes in middle ears of the younger patients so treated by repeated medical "abortions."

Surgery is an individual proposition. The listless child with vague ear symptoms and X-ray evidence of mastoid disease responds to surgery very well.

Resolution of the acute mastoiditis may occur quite naturally when the drum perforates, and constant drainage is established. There has been permanent damage.

Is there such a saving in health, time or money to the patient given repeated courses of antibiotics, as compared to a clean-up trip to the operating room? There is no saving to those who have had several bouts of antibiotics and long periods of malaise, vague abdominal discomfort, diarrheas, dementias and dermatitis. These are the patients who finally wonder if surgery is not the answer, and how disappointing it must be for such a patient to have his otolaryngologist suggest further antibiotic therapy.

Modern surgery can compete safely with medicine alone in every aspect of mastoiditis. We may decry this reliance on antibiotics and the pernicious therapeutic philosophy outlined by Wesley Spink[11] that : (quoted verbatim)

1. Antibiotics are highly effective for many common infections.
2. That these are readily available, inexpensive, easy to administer and non-toxic.
3. With a febrile patient, cause unknown, give an antibiotic.

Ask any mother whose child had repeated bouts of middle ear infection, nosedrops, tonsillectomy, allergy treatments, radium treatments, X-ray treatments, *ad infinitum*, how much happier ultimate surgery has made her and her child.

The most important surgical justification in the operating room as to need for surgery is probably the color of the lateral sinus and whether or not it is lusterless and violaceous.

Only *anesthesia* need complicate surgery, and this should be open drip ether for children and Flagg administration for the adult, with change to the smaller can after thirty minutes, to prevent stagnation and rebreathing. With or without burrs, and regardless of route, the surgical procedure of simple mastoidectomy is of great value.

CONCLUSIONS.

It is doubtful whether the incidence of patients requiring or meriting careful consideration of the need for surgery has been materially reduced by antibiotic therapy.

Many children are needlessly becoming hearing problems.

The antibiotic, which has its justly earned place in the therapeutic armamentarium, should be used as an adjunct to rather than a substitute for surgery in lessening the dangers of operating through an infected field and preventing complications.

Only surgery can drain the tip or remove the dense central plate blocking the aditus.

Though symptomatology has been minimized by the administration of antibiotics, it is doubtful whether the true pathology has been greatly altered or reversed thereby.

In the patient with two or more recurrent bouts of otitis media in the same ear within a year or less, conservative procedures having been previously carried out, and with X-ray evidence of damage present, one should consider surgery.

Once mastoidectomy has been decided upon and procedure in progress is completed, the surgically treated mastoid should clear up quite satisfactorily without the administration of antibiotics. If these are administered, they should be discontinued as soon as possible, to observe the natural changes in temperature and white count.

We, as otolaryngologists should approach the serious and confusing problem boldly rather than abandon a tried and true surgical principle.

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TUBEROUS SCLEROSIS. A CASE REPORT.

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The purpose of this report is to add to the literature an example of a rare syndrome and to call the otologist's attention to points wherein he might be of help in the diagnosis.

Tuberous Sclerosis is characterized clinically by the triad of mental retardation, epileptic seizures, and a peculiar type of cutaneous lesion called sebaceous adenoma. It has been referred to as one of the congenital ectodermoses and is frequently associated with Von Recklinghausen's disease. Other congenital neoplastic formations, such as tumors of the kidney and retina, are frequently associated, since it is a blastomatous ectodermal defect.[1]

A comprehensive review of the literature is difficult because of the association of tumors of other origins; however, it is interesting to note that significance of the histological alterations was first appreciated in 1912, when Bundeschuh gave us complete studies based on the embryological concepts of Ranke. Different aspects of the triad are emphasized by different authors; however, our primary interest is centered on the cutaneous lesion. In 1891 Pringle[2] reported a case of congenital adenoma sebacium citing that the tumors growing on the face were small papillary growths varying in color from pale to dark red. They ranged in size up to one cm. and had a butterfly distribution over the nose and cheeks.

E. L. S., a 24-year-old white male, reported to this clinic complaining of intense itching, pain, and discharge of the ears. This proved to be a rather severe case of diffuse external otitis which responded quickly to treatment. The most notable findings were many small papillary tumors arranged in a butterfly facial distribution and filling the external canal. This observer's curiosity was not aroused sufficiently at that time to remove one of the tumors for biopsy. A few weeks later a local urologist[3] reported to us that he had removed a large hamartoma of the kidney and that the diagnosis of tuberous sclerosis was considered. The patient revealed that he had had Jacksonian type seizures intermittently since childhood. At this point, the pathology report was obtained on one of the ear tumors. It is recorded:

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"Microsection of the biopsy removed from the region of the right ear reveals moderate increase in the number of sebaceous glands with increased keratinization in follicles in these glands. Frequently the follicles are dilated and contain desquamated, keratotic debris. The necks of the sebaceous glands are lined with stratified squamous epithelium. About these glands there is a moderate chronic inflammation. It is much more marked just beneath the epidermis than about the glands. Even in this location it tends to be rather focal in distribution. There are no changes present that would suggest malignancy and as far as can be determined, the primary change is that compatible with sebaceous adenomas."

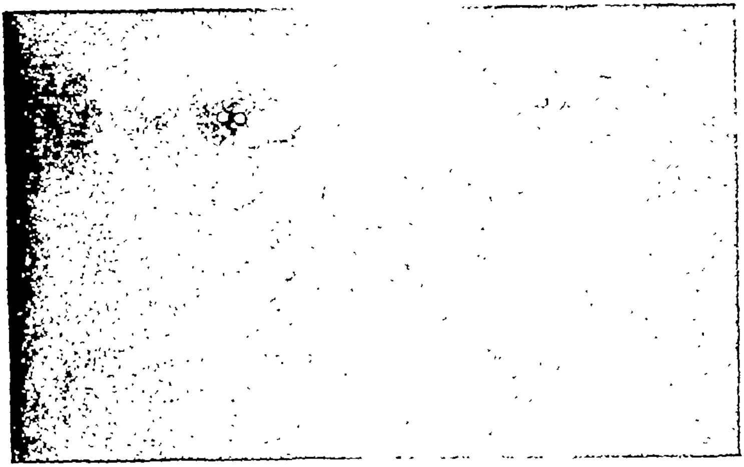

Fig. 1.

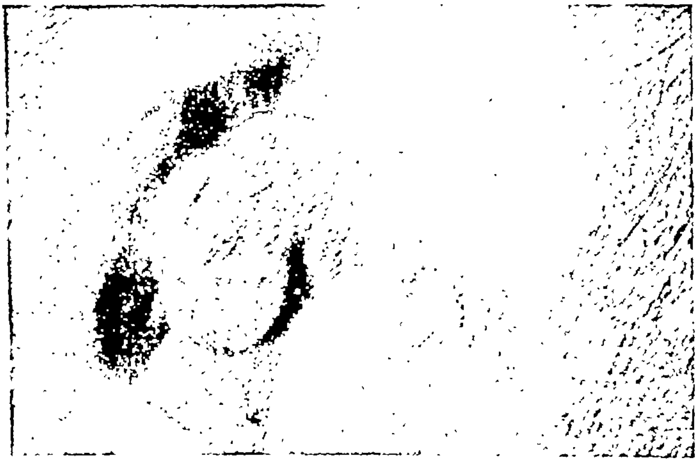

Fig. 2.

In summary, a brief review of the literature and an illustrative case of tuberous sclerosis is presented. It behooves the otolaryngologist to delve deeper into the history of patients presenting small fibromatous tumors of the external ear.

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POSTGRADUATE COURSE IN OTOLARYNGOLOGY

The Department of Postgraduate Medicine of the University of Michigan Medical School announces the Otolaryngology Conference to be given at the University Hospital, Ann Arbor, Michigan, April 15-17, under the direction of Dr. A. C. Furstenberg, Chairman of the Department of Otolaryngology, University of Michigan Medical School.

Guest Lecturers: Dr. J. A. Hilger, St. Paul, Minn.; Dr. H. P. Schenck, Philadelphia; Dr. Paul Holinger, Chicago; Dr. K. M. Day, Pittsburgh; and Dr. Lawrence R. Boies, Minneapolis, Minn.

Resident Lecturers: Dr. A. C. Furstenberg, Dr. J. H. Maxwell, Dr. J. E. Magielski, Dr. Thomas Francis, Dr. Jerome Conn, Dr. E. R. Harrell, Jr., Dr. R. B. Sweet, Dr. H. E. Sloan, Jr., and Dr. Irving Blatt.

For further information, address: Dr. H. H. Cummings, Chairman, Department of Postgraduate Medicine, University Hospital, Ann Arbor, Michigan.

FROZEN SECTION BIOPSY IN PERORAL ENDOSCOPY.

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In the last years peroral endoscopy has become a routine procedure of examination. The specialist is able to make a very satisfactory and accurate examination of the larynx, trachea, bronchi and esophagus, but a large number of lesions located in these structures can not be actually diagnosed without the help of peroral endoscopy. Benign or malignant tumors, inflammatory lesions can be visualized, examined and a biopsy taken for the necessary histologic diagnosis. This undoubtedly is one of the greatest advantages of peroral endoscopy. If, due to circumstances, the biopsy in a few cases fails in the first attempt to bring out sufficient material for histologic diagnosis, it is necessary to repeat the endoscopic examination; and in a large number of cases this is not very well accepted by the patients.

To avoid such inconvenience at Casa de Saude Sao Miguel, we have been obtaining very good results during the endoscopic examinations by using routinely the frozen sections technique for histologic diagnosis of the biopsy material. The tissue obtained by the biopsy forceps is immediately taken to the pathologist, working in a laboratory close to the operating room, where it is sectioned, stained and examined under the microscope. The histologic diagnosis by this method is given to the endoscopist in a period of time varying from two to three minutes. If the biopsy is negative or insufficient, the endoscopist still has time and opportunity to take one or more pieces of tissue from the lesion without further inconvenience or risk to the patient.

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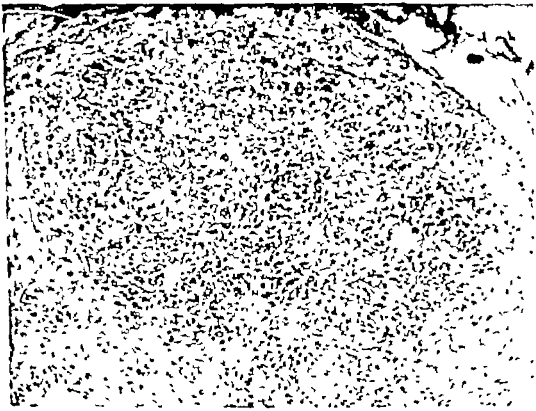

Fig 1 Larynx

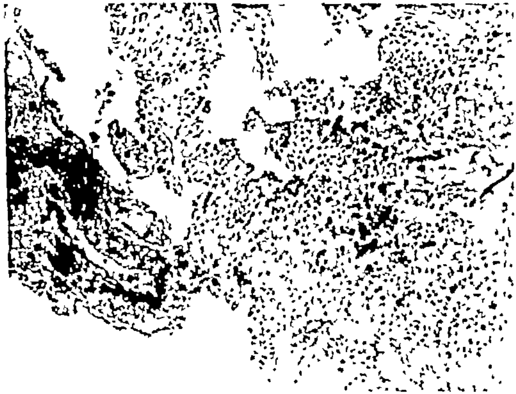

Fig. 2 Bronchus

By this method, at the Casa de Saude Sao Miguel, from 1950, we had an opportunity to examine and diagnose cases of lesions located in the larynx, trachea, bronchi, esophagus and base of the tongue.

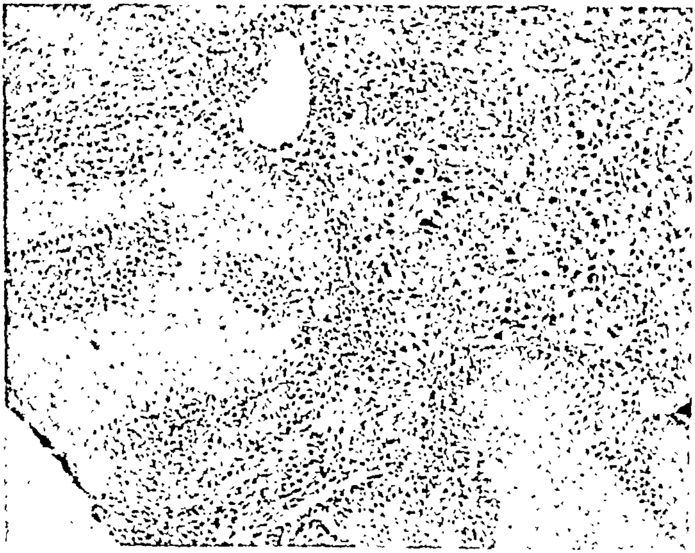

Fig. 3. Esophagus.

The histologic diagnosis and location of the lesions are shown in Table I.

In three cases of our series, two or more successive biopsies were necessary to reach a correct diagnosis. In these cases the delay of a few minutes caused no inconvenience to the patients. In 31 cases, the first piece of tissue taken by the biopsy forceps was sufficient for an immediate, correct histologic diagnosis. In three cases, two fragments were necessary, and in two cases, three fragments were necessary.

Table I.

Location	No. of Cases	Benign Tumors or Inflammatory Lesions	Malignant Tumors
Larynx	5	2	3
Bronchi	16	6	10
Esophagus	14	1	13
Base of Tongue	1	–	1
	36	9	27

TECHNIQUE.

The tissue obtained by biopsy forceps, is immediately taken to the pathologist. We use a frozen microtome to make the sections. We emphasize that with little practice small pieces of tissues can be very well sectioned in the frozen microtome, giving as good and as satisfactory sections for diagnostic purposes as large fragments. We prefer the Terry stain, or "neutralized polychrome methylene blue," which in our opinion has the following advantages:

1. Fixation is not necessary.
2. It is a vital stain and stains in few seconds.
3. It is extremely useful for diagnosis of malignant cells.

The sections so stained are covered by a coverslip and examined under the microscope. The same block of tissue is afterwards run through 10 per cent formalin, alcohol, xylol, etc., imbedded in paraffin, sectioned on the microtome, stained by routine hematoxylineosin, and the slides are kept in the laboratory files after being checked. Even the original sections can be stained afterwards by hematoxylineosin, after the necessary steps through 10 per cent formalin, alcohol, xylol, etc.

We consider the method, as described above, absolutely reliable. It is not necessary to bring out the advantages of this simple procedure to the endoscopist, surgeons and patients.

SUMMARY.

The authors advise the use of the frozen sections method for histological diagnosis during endoscopic examinations. They believe this method is absolutely reliable giving results identical to other methods, (paraffin sections), with the advantage of shortening the time necessary for histologic diagnosis.

In our cases, in which biopsy was possible, no patients left the operating room without diagnosis.

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PERCEPTIVE DEAFNESS TREATED WITH VITAMIN B-COMPLEX AND AMINO ACIDS—100 CASES.

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Previously the failure of Vitamin B-Complex to influence the course of progressive perceptive deafness was reported.[1] Since then, in addition to the B-Complex, amino acids and histamine acid phosphate have been given over a 10-year period, and herewith is presented a report on 100 patients with perceptive deafness, tested before treatment was started and again an average of 9.79 months afterwards.

Since all these patients had the same type of deafness and were treated much the same, composite audiograms are the only accurate measures of the effect of therapy. While isolated cases reported improvement after treatment, either regarding tinnitus, which was a primary symptom, or the hearing loss, it seemed better to depend upon the audiograms which are precise and the only sure measure of curbing the indiscriminate use of unestablished forms of therapy.

Perceptive deafness is not an uncommon accompaniment of middle age, and occurs in various occupations in which there is prolonged exposure to intense noise. It may follow the use of drugs such as streptomycin or quinine, and is seen after high fevers or as a complication of meningitis. Since World War II its incidence is greater, due to the increase in the number of men and women working in heavy industry and serving in the armed forces, and the otologist now sees more young individuals with occupational deafness.

That noise is recognized as an occupational hazard, is indicated by cases on record in which damages were recovered by individuals deafened as a result of their employment. Industrial concerns and the armed services should obtain audiograms (other tests for hearing are not sufficiently exact) on

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all employees, and on those in service, at the time of hiring or the beginning of service, and again at the termination of employment or of service.

While deafness is recognized as a hazard in operation of motor vehicles in 36 States and the severely deafened might be forbidden to operate motor cars in those States, this restriction is probably too seldom enforced. It should be a national requirement that the hearing acuity of applicants for drivers' license be at least roughly tested with whispered or spoken voice before a license is granted.

Perceptive deafness is usually progressive, as is suggested by the composite audiograms on five untreated cases observed over an average period of two years, in which there was an increased hearing loss of 12.8 per cent binaural. Spontaneous recovery from perceptive deafness is remote, and "it is doubtful if any organ of the human body has as little power of recuperation as the organ of hearing."[2] In the 100 cases presented here there was increased hearing loss of 2.5 per cent binaurally, after 9 months, and while treatment did not result in improved hearing, it may have slowed the progress of the deafness; although probably not enough to justify the expense and effort expended.

Williams and associates,[3] by restricting the thiamin intake to an average of 0.35 mg. daily for 110 days were able to show development of polyneuropathy, consisting of defects of the sensory nervous pathways, loss of tendon reflexes and paralysis of the muscles of the legs, which after many weeks responded to large doses of thiamin. They concluded that the polyneuropathy was the result of late severe, rather than early mild, deficiency of thiamin. No mention of the organ of hearing is made. Kopetsky[4] was unable to help cases of deafness with thiamin alone, but advocates the use of the whole B-Complex, amino acids, trace metals, thyroid and pituitary extract, histamine desensitization and other measures.

The treatment used in the cases reported here consisted of large doses of amino acids and B-Complex orally, B-Complex parenterally, and histamine intravenously. When occasionally the amino acid product in liquid form was poorly tolerated, it was given in powder form. Most patients were given at least two drugs simultaneously, and were also put on a diet rich in Vitamin-B. The results were disappointing.

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BRONCHOESOPHAGOLOGY COURSE.

The next Bronchoesophagology Course to be given by the University of Illinois College of Medicine is scheduled for the period, March 22 through April 3, 1954. The course is under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

MALIGNANT MELANOMA OF THE NASAL SEPTUM

FRED W ALEXANDER, M D ,

Cleveland, Ohio

The purpose of this paper is to report what I believe is the twenty-fifth case of malignant melanoma of the nasal septum Of all malignant melanomas of the nasal cavity and sinuses, there are only 93 cases mentioned in the English literature. In many of these cases the site of origin is vague, but 24 cases are definitely cited as originating on the nasal septum Pipia, cited by Grace,[1] states that these tumors never arise in the superior portion of the nasal membranes, but always arise in the lower portion of the nasal septum and middle or inferior turbinate I find that this is essentially true in the cases reported to the present date, though Pipia made his observation in 1927

CASE REPORT

On March 6 1948 a white 59 year-old female presented herself with the complaint of being unable to breathe through her nose for four months. Past history revealed that she had seven nasal polypectomies in the last 25 years In 1940 she had an operation on her forehead for what may have been an osteomyelitis of the frontal bone Nose bleeds were intermittent in the past, and because of these and/or mild hypertension she was under mild sedation and on a severe diet that had caused her to lose 20 pounds A goiter had been present for 20 years and other than the nasal polypi the patient stated that she had been in good general health.

Examination revealed a cruciate incision of the forehead in the mid line with a slight depression of the outer table of the forehead No glands were palpable in the neck A large movable very hard goiter presented itself in the suprasternal notch The left naris contained many polypi with complete obstruction of the airway The right naris was also obstructed with many large nasal polyps and showed signs of recent bleeding The polypi in the right naris protruded into the vestibule bled easily when touched and were of a normal fish flesh color Mouth tongue and pharynx were normal. There was much mucoid discharge in the nasopharynx, but the posterior naris Eustachian tube orifices and palate were normal The patient was edentulous with full upper and lower dentures. The larynx was normal in appearance and function Large tonsils were present, but not infected Both ears were normal with intact drums and normal hearing on audiometric examination Sinuses were clear to transillumination Chest was clear to auscultation and percussion heart was normal in size and sounds blood pressure 180/90 No masses or glands palpable anywhere on body Chest X rays normal. Weight 118 pounds.

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Course: On March 8, 1948, the patient was admitted to St. Luke's Hospital, and multiple nasal polyps were removed from both nares and ethmoid sinuses. During the course of the operation a darkly pigmented livid-brown sessile 1 cm. mass was noticed on the right side of the nasal septum located 2½ cm. from the mucocutaneous junction opposite the anterior end of the middle turbinate, 2½ cm. from the floor of the nose, in the mid-septum. This mass was raised from the surface slightly and bled when touched. Because of its unusual appearance the mass was biopsied and the base obliterated with actual cautery. The mass did not seem to extend below the mucosa, and was not connected to the cartilaginous septum. The pathologist reported the biopsy as malignant melanoma, and the other excised material as nasal polyps. On March 12, 1948, under cocaine anesthesia, with a Bovie knife the entire nasal septum was removed *enmasse*, giving a wide 2½ cm. margin of normal tissue between the incision and the tumor edges. This was examined by the pathologist in a very careful manner, and no evidence of tumor could be found on the resected portion of the septum or in the area of cauterization at the site of the tumor. Postoperative recovery was uneventful, and the patient was discharged, but was kept under observation at frequent intervals.

Six months later, September 2, 1948, following an acute upper respiratory infection, the patient developed swelling of the forehead, and was admitted with a diagnosis of acute osteomyelitis of the frontal bone on the right. This was confirmed by X-ray studies, and September 3, a radical removal of both frontal bones and obliteration of the frontal sinuses was performed. Microscopic examinations of the tissues removed revealed osteomyelitis and infection without evidence of any tumor. The nose was clear of tumor, and no masses or suspicious areas could be seen on any of the visible surfaces of the mucosa of the nose, septal perforation, turbinates, or nasopharynx. Recovery was uneventful, and patient was kept under observation at monthly intervals until September, 1949. During the year she maintained a weight of 140 pounds.

Nineteen months after the discovery of the initial lesion of the septum, September 24, 1949, the patient complained of slight bleeding from the throat and mild obstruction of breathing through the left nostril. On examination a small darkly mottled mass about 3 cm. in diameter, which bled easily on palpation, was seen protruding from the left side of the vault of the nasopharynx. This mass was removed under local anesthesia and proved to be malignant melanoma. No glands were palpable in the neck or elsewhere. X-ray survey of the neck, sinuses, skull, and chest were normal except for old operative defects. Two months later the mass in the vault of the nasopharynx again became noticeable but was not touched until February 6, 1950, when the mass had become pedunculated and started to bleed. The mass was then removed by simple local excision under general intratracheal anesthesia, and again proved to be malignant melanoma. The patient's weight remained at 148 pounds, and no evidence of tumor could be seen in the nasopharynx or elsewhere until June 24, 1950, when a small mass appeared in the vault of the nasopharynx over the right eustachian tube. This was watched until September, 1950, when a large node appeared high in the left side of the neck and another node in the right axilla. No therapy was given, but observation continued until February 27, 1951, three years after discovery of the initial lesion, when the mass was again removed from the nasopharynx because of bothersome bleeding. No local recurrences could be seen in the nose or about the nasal septal perforation following this operation. The large node in the left side of the neck and in the right axilla gradually disappeared completely, after being present for 22 months. The patient weighed 155 pounds on June 30, 1951.

In July, 1951, the nasopharyngeal mass recurred to an extent with sufficient bleeding to require removal again for the fourth time, and the nasopharynx remained clean until October 18, 1952, at which time a severe respiratory infection caused the right side of the forehead to swell and the right eye to close, but the infection subsided under antibiotics without further surgery At that time a small mass was noticed recurring in the right nasopharynx, but did not cause sufficient symptoms or bleeding until further extension over a gradual period of time began to involve the right posterior naris and posterior ethmoid area This gradually spread into the right ethmoid sinus area until bleeding became a problem Again, and for the fifth time, the mass was removed from the nasopharynx and right ethmoid area on December 12, 1952, four years after initial discovery Despite these many vicissitudes, the patient remained well and active physically between bouts, with an average weight of 148 pounds, until January, 1953, when the right anterior ethmoid area began to bleed from a recurrence of the mass in that area The nasopharynx remained clear of tumor On March 27, 1953, a large mass was removed from the right ethmoid area under general anesthesia and bleeding was severe, being controlled with difficulty At this time it was thought that the cribriform area had been involved, but the patient quickly recovered and gained weight up to 150 pounds and went about her business with vigor until June, 1953

June 9 1953, five years and four months after the initial lesion's discovery, the patient complained of persistent headache of vague location and developed more bleeding from the right ethmoid area The tumor from the ethmoid sinus extended up into the cribriform area and upper septum Bleeding was controlled with intermittent packing for a time, as it was thought we were nearing the end of the road and that further surgery would be futile, however on June 27, 1953, bleeding became so severe that the patient was admitted and the nose packed On July 1, 1953, bleeding became uncontrollable and patient was taken to surgery and the easily accessible portions of the tumor were removed from the right ethmoid and septal area, where it was obvious that the tumor had penetrated the cribriform area toward the brain. Postnasal packs and heavy vaseline and cellulose packing to both nares controlled the bleeding To our surprise we were able gradually to remove all packing, and the patient left the hospital by the tenth postoperative day The patient continued to have headaches, but not too severe to prevent her getting about and doing light work until August, 1953, at which time bleeding gradually became more pronounced as the tumor in the right ethmoid and cribriform area grew more massive On August 29, 1953, for the eighth time, the patient went reluctantly to the hospital because of severe bleeding, and a huge mass of tumor was removed from the right ethmoid and cribriform area The cribriform area was markedly involved and its line of demarcation poor Much necrosis and infection was apparent in the area. The area was packed to control hemorrhage and the patient did well for two days and packing was removed without bleeding Three days postoperatively the patient became disorientated and developed a high fever, without stiff neck, and we suspected brain abscess; however, despite supportive treatment and antibiotics, the patient did not rally, and on the tenth postoperative day showed 2000 cells in the spinal fluid with definite signs of meningitis. This progressed rapidly with severe pulmonary edema and death on the twelfth postoperative day, September 10, 1953, five years and six months after discovering the malignant melanoma of the nasal septum

Autopsy report summary is as follows Malignant melanoma of the nasal septum with direct extension to the nasopharynx, paranasal sinuses, and the base of the brain The brain showed a mass 2 8 x 3 1 cm., with diffuse mottling of brownish black pigment extending into the lower portions of the frontal lobes 3.5 cm. with a necrotic area involving the center of the tumor. No distant metastases were found in the lymphatic or other systems.

Figure 1 shows a typical section of the malignant melanoma.

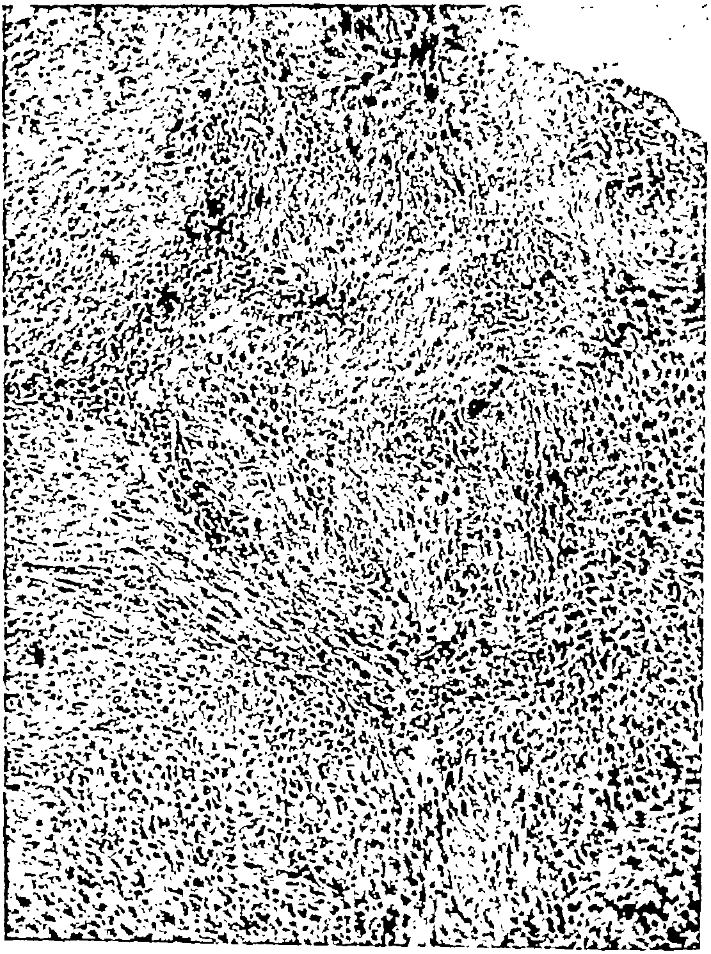

Fig. 1. Typical high-power section of the malignant melanoma, from the nasal septum.

COMMENTS.

Adequate review of the literature and cases reported up to 1947 is given by Grace.[1] Alsup[2] gives the subject good coverage through 1949. These two papers cover all malignant melanomas of the nasal cavity and adjacent sinuses reported in the literature, through 1949, *i.e.* 69 cases. Only 13 cases appeared in American literature until 1949. Since 1949 there are 24 other cases reported in the English literature as follows: Schoolman and Anderson,[3] six cases in negroes; Morris and Horn,[4] one case in a negro; Morey,[5] one white case; Capps and Williams,[6], one case; and T. S. Stewart[7], 15 cases; making a total of 93 cases involving the nasal cavity and sinuses. Only 24 of these cases are definitely located on the nasal septum. Many are poorly located as to exact site, but are referred to as in the nasal cavity or sinuses. In general, the most common sites are the lower anterior half of the septum, anterior halves of inferior and middle turbinates, inferior meatus, and floor of the nose. Involvement of the sinuses is much less common than in carcinoma, which shows up in the middle meatuses. Several melanomas are reported that seem to originate far back in the floor of the nose, visible only with posterior rhinoscopy.

Nearly all cases are associated with nasal polypi, and are discovered during operation for removal of the latter. In a few cases the tumor was discovered by the careful scrutiny of a dark colored or freckled area that bled easily on touch. Bleeding and distention of the naris by an intranasal mass is a very common early symptom. It is obvious from reported cases that many of the tumors are not recognized from the appearance of the polyp, or sessile mass, and that the tumor was not recognized until it rapidly recurred with disastrous results. Hence it follows that in all nasal polypectomies the tissues removed should be subjected to miscroscopic pathological examination. From the numerous recent reports, it is also obvious that malignant melanomas of the nasal cavity are far more common than we suspect. Stewart points out that 4 per cent of 300 malignant tumors of this area were malignant melanomas, 80 per cent carcinomas, and the rest malignant tumors of other types.

The prognosis in these tumors is poor for ultimate survival, but 5 per cent may survive from two to five years. Once the lesion has been operated upon or disturbed, the growth seems to accelerate and may metastasize to any area, or recur only locally at a rapid rate; however, it is a fact that if they survive the first two years, they may go several more years (up to five years) without too much difficulty. Strange as it may seem, the cases that survived the longest were those having frequent local excisions of recurrences. Alsup reports a case that lasted seven years and three months. The prognosis of these tumors originating on mucous membranes is poorer than those occurring in the eye or skin, where long periods may elapse between the appearance of the "latent metastases." In tumors originating on the mucosa of the nasal cavity the periods of quiescence of the latent metastases are short, the longest of the recorded cases being three years.

Treatment by radiant energy (X-rays, radium, etc.) of tumors of this type, and in these areas, is of no value at the present time. This is true of melanomas elsewhere. Packs'[8] principle of "dissection in continuity" is still the procedure of choice in these tumors, and should be practiced despite the difficulty encountered with these cases due to their position in the nasal cavity. This principle should also be followed despite the fact that recorded cases that have had repeated local excisions survive the longest. I believe that radical excision early would increase the extremely poor survival rate, but that once extension has taken place from inadequate removal, no matter how radical, all hope is gone.

SUMMARY.

A case of malignant melanoma of the nasal septum, twenty-fifth in the literature, is reported.

Malignant melanomas are more common in the nasal cavity than is generally recognized.

Radical surgical excision should be practiced at the earliest possible moment if any hope of ultimate survival is to be obtained in these otherwise fatal cases.

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10515 Carnegie Ave.

THE AMERICAN OTORHINOLOGIC SOCIETY FOR PLASTIC SURGERY, Inc.

The regular and scientific meeting of The American Otorhinologic Society for Plastic Surgery, Inc., will be held on Saturday, March 27, 1954, at Hotel Statler, Boston, Mass.

Following is a schedule of the program:

2:00 P.M.—"The Management of Cleft Palate Cases Involving the Hard Palate, So As Not to Interfere with the Growth of the Maxilla."—Dr. Fred Squier Dunn (by invitation). Discussion—Dr. Raymond Rosedale, M.D., M.Sc. (Med.)

3:00 P.M.—"Surgical Management of Complete and Incomplete Bony Atresia of the Posterior Nares."—Dr. Henry Beinfield. Discussion—Dr. Matthew Ersner.

4:00 P.M.—Display of Special Exhibits.

6:30 P.M.—Cocktails and Dinner.

8:00 P.M.—"The Management of Congenital Cleft Lip and Associated Nasal Deformities."—Dr. Douglas Parker (by invitation).

9:00 P.M.—"Round Table Discussion on Otorhinological Problems." Moderator—Dr. Nathan Bolotow.

For further information write Dr. L. J. Feit, 66 Park Ave., New York 16, N. Y.

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February 1, 1954.

Acousticon Models A-17, A-180 and A-185.
Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 1, New York.

Auditone Models 11 and 15.
Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67 and 70.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac Model M; Mono-Pac Model "Lyric"; Mono-Pac Model "Rhapsody."
Manufacturer: Beltone Hearing Aid Co., 2900 West 36th St., Chicago 32, Ill.

Cleartone Model 500; Model 700; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Model D-1; Dahlberg Junior Model D-2; Dahlberg Model D-3 Tru-Sonic; Dahlberg Model D-4 Tru-Sonic.
Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Fortiphone Models 19-LR; 20A; 21-C and 22.
Manufacturer: Fortiphone Limited, Fortiphone House, 247 Regent St., London W. 1, England.
Distributor: Anton Heilman, 75 Madison Ave., New York 16, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.
Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.
Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico Model J; Maico Top Secret Model L; Maico Maxitone.

Manufacturer Maico Co., Inc., 21 North Third St., Minneapolis, Minn.

Micronic Model 303; Micronic Model "Mercury"; Micronic Star Model

Manufacturer Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Classic Model T9; Microtone Model T10; Microtone Model T612.

Manufacturer Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn., Minneapolis 9, Minn.

National Ultrathin Model 504, National Vanity Model 506

Manufacturer National Hearing Aid Laboratories 106 So 7th St., Philadelphia 6, Pa.

Normatone Model C and Model D-53

Manufacturer Johnston Hearing Aid Mfg Co, 708 W 40 St., Minneapolis, Minn.

Distributor Normatone Hearing Aid Co., 22 East 7th St., St. Paul (1), Minn.

Otarion Models B-15 and B-30, Otarion Models F-1, and F-3; Otarion Model G-2, Otarion Model G-3; Otarion Model H-1; Custom "5."

Manufacturer Otarion Hearing Aids, 4757 N Ravenwood, Chicago 40 Ill.

Paravox Model D, "Top-Twin-Tone"; *Model* J (Tiny-Mite); Paravox Model Y (YM, YC and YC-7) (Veri-Small)

Manufacturer Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio

Radioear Model 62 Starlet; Model 72; Model 82 (Zephyr)

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Distributor Radioear Corp 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16 Pa.

Silvertone Model H-16, J-92, Silvertone Model P-15

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Solo-Pak Model 99.
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Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966; Sonotone Model 977; Sonotone Model 988.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

Televox Model E.
Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 953; Telex Model 1700.
Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.
Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster; Model Cameo.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal; Zenith "Regent."
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Manufacturer: Audivox, Inc., 123 Worcester St., Boston 18, Mass.

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Otarion Model C-15; 1 transistor, 2 tubes, and 2 batteries (A & B).
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Sonotone Model 1010; 1 transistor, 2 tubes, and 2 batteries (A & B).
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Telex Model 954; 1 transistor, 2 tubes, and 2 batteries (A & B).
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THE ELECTROLYTES OF THE LABYRINTHINE FLUIDS.[*]

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The endolymph and perilymph of the inner ear have been the subject of speculation for many years. Theories concerning their origin and relation to each other still lack definite proof. Anatomical evidence points toward two fluids in separate compartments. Experimental evidence has been conflicting as to the permeability of Reissner's membrane and other parts of the membranous barrier. Little is known about their normal composition. From existing analytical data it appears that the labyrinthine fluids differ but slightly from spinal fluid and each other.

An extensive analysis of the pooled ear fluids from freshly killed sharks (Scolioidontus laticandus) was made by Kaieda[1] in 1930. There have been several attempts to repeat the analysis of fluids from living mammals. Table I gives values reported by previous investigators. Only those results from Kaieda's analysis that are pertinent to the present studies are included.

Kaieda[1] showed that shark perilymph had a slightly higher organic content than endolymph; endolymph had a higher in-

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organic content, as shown by its higher electrolyte concentration. Ledoux and his associates analyzed dog and cat labyrinthine fluids for a number of constituents. They observed small but consistent differences in the osmotic pressure of endolymph, perilymph, cerebrospinal fluid, and plasma.[2,3] Ledoux[4] reported that the protein content of cat perilymph is twice that of endolymph, and almost ten times that of spinal fluid. Waltner and Raymond[5] found much less protein in human perilymph (Meniere's disease). Szasz[6] gives a higher value for the refractive index of spinal fluid compared to perilymph in the dog, while Ledoux[7] gives a lower value. Values given by Ledoux[4,8] for urea N, chlorides, and reducing substances show negligible differences in the three fluids. Waltner and Raymond[5] reported that a single sample of human perilymph had almost the same sodium concentration as spinal fluid.

TABLE I.
DATA OF OTHERS ON ENDOLYMPH, PERILYMPH AND SPINAL FLUID.

	Animal	Perilymph	Endolymph	Spinal Fluid	Author
Viscosity (Relative to water)	Pigeon	1.7	2.9		Rossi[10]
Refractive Index					
$N_D^{17.5°}$	Dog	1.33515		1.33527	Szasz[6]
$N_D^{22.5°}$	Dog	1.3349	1.3347	1.3342	Ledoux[7]
$N_D^{22.°}$	Cat	1.33495	1.33455	1.33435	Ledoux[7]
Osmostic Pressure					
Relative to plasma	Cat	1.006	1.004	0.998	Ledoux[3]
NaCl equivalent in m.Eq./L	Cat	179	181	174	Aldred, Hallpike and Ledoux[2]
pH	Cat	7.87	7.82	7.45	Ledoux[8]
Electrolytes					
Sodium m.Eq./L	Shark	231	542	253	Kaieda[1]
Potassium m.Eq./L	Shark	14	24	14	Kaieda[1]
Chlorides m.Eq./L	Shark	245	464	231	Kaieda[1]
	Cat	152	158	151	Ledoux[8]
Protein mg. per cent	Cat	268	118	31	Ledoux[4]
	Human*	27		13	Waltner and Raymond[5]

* Meniere's cases, Average of three cases.

Graf and Poretti[11] (1950) measured the rate of entry of radioactive Na into the labyrinthine fluids of the guinea pig. They found that the radioactive Na in the perilymph increased from 20 per cent of the spinal fluid concentration at 37 minutes to 82 per cent at six hours. During this same interval, the concentration of the tracer in endolymph increased only from 5 per cent of the spinal fluid level to 9 per cent. There was little change in this ratio between one and six hours. The low initial levels in endolymph might indicate a slow exchange of the sodium in endolymph with perilymph or plasma; however, the failure of the level to rise appreciably after an hour suggests another possibility: that there may be a low total sodium concentration in endolymph.

To explore this possibility and to supplement the incomplete knowledge of labyrinthine fluid composition, the endolymph, perilymph, spinal fluid and plasma of the guinea pig have been analyzed for Na, K, Cl, and protein. Because of the changes which have been shown to occur in Na and K concentrations during propagation of the nerve impulse, these ions were of special concern. It has been found that endolymph differs strikingly from perilymph and spinal fluid in regard to Na and K.

SURGICAL TECHNIQUE.

Healthy, dark colored guinea pigs were used. They were of 250-350 gm. weight (6-8 weeks of age) with good hearing as shown by the pinna reflex. The animals were anesthetized with "Dial" (diallylbarbituric acid with urethane, Ciba), 0.5 ml. per kg., and 0.5 ml. of 1 per cent procaine.

Perilymph. The bulla was exposed and an opening made in it by dental drill. The tympanic membrane was carefully displaced from about half of its bony attachment in order that the lateral portion of the bulla, including the external ear canal, could be removed without undue tension on the ossicular chain. Both round and oval windows were then fully accessible. The round window membrane was cleaned of any tissue fluid by a wisp of cotton or small piece of gelfoam. The membrane was then pierced by a pyrex micropipette in a micromanipulator, and perilymph was withdrawn from the scala tympani of the cochlea. The pipette was made of 3 mm. tubing drawn down to 0.3 mm., 1 cm. from the tip, and 0.1 mm. at the tip. The pipette remained in the perilymphatic space for a minimal period (30-40 seconds) to avoid the possibility of contamination through the cochlear aqueduct.

Utricular Endolymph. The malleus and incus were next removed and the facial nerve unsheathed and displaced. The stapes was pulled up out of the oval window and the opening enlarged to give better visibility of the utricle. The vestibule was kept free of perilymph by intermittent suction at the round window. The utricle was pierced by a micropipette in a micromanipulator. As the endolymph was withdrawn, the utricle visibly collapsed. Although care was taken to avoid the capillaries of the round window membrane and utricle, this was not always possible. If more than a trace of blood was visible, the sample was discarded. The samples obtained ranged from 0.2 to 1 μl. (.0002 to .001 ml.).

Cochlear Endolymph. The cochlea was exposed as described above. In two animals (Nos. 89 and 98) the basilar membrane was pierced at the round window to enter the cochlear duct. The method has been described by Aldred, Hallpike and Ledoux[2] and by Graf and Poretti[11]. The vestibule and cochlea were cleared of perilymph. Constant suction, or a piece of gelfoam at the cochlear aqueduct was necessary to prevent the entry of spinal fluid. The technical difficulties could not be adequately overcome, and the method was abandoned, although a few valid specimens were obtained.

In other animals the cochlear duct was entered in the second or third turn through the stria vascularis. This was exposed by making a small hole in the bony cochlear wall with dental burr and steel pick. The round window was opened and the stapes loosened in the oval window to reduce the flow of perilymph through the artificial fenestra. A small piece of gelfoam was placed on either side of the hole. This appeared to absorb adequately the perilymph, and the outer layer of the stria vascularis seemed to be free of fluid. The stria vascularis was pierced by a small micropipette (40 microns outside diameter). A small amount of fluid immediately ran up by capillary attraction. Usually nothing more could be withdrawn by suction, and rarely could more than 0.4 μl. of fluid be obtained.

Cerebrospinal fluid was removed from the fourth ventricle. In some animals blood was removed from the heart for serum analysis.

All pipettes were cut short immediately upon withdrawal, sealed at both ends with paraffin or beeswax and refrigerated until surgery was completed. Most of the animals were perfused through the aorta with Heidenhain-Susa fixative, and the operated ear was removed, dehydrated and embedded in celloidin. Serial sections were stained with hematoxylin and eosin.

Sections from pigs which had furnished utricular endolymph showed that the medial wall of the utricle had been broken in only one animal of 22 examined. The ears from the remaining animals showed a large hole in the lateral wall of the utricle near the macula, where the pipette had entered. In three cases there was microscopic evidence of injury to the vestibular cochlear duct during the enlargement of the oval window. In four instances there was a small tear in the saccule at the point where it is attached to the utricle. As considerable pressure was sometimes necessary to pierce the utricular membrane, this tear was probably made as the pipette entered the utricle. In four cases sections of the vestibular cochlear duct were incomplete, due to faulty technique. The remainder showed the membranous labyrinth to be completely intact except for the hole made by the pipette.

Histological examination demonstrated less success in the collection of uncontaminated cochlear endolymph. On the basis of the examination of serial sections and the operative protocols, the animals were divided into three classifications according to the degree of confidence in the samples (Table IV). Data from one animal were discarded, because sections revealed puncture of Reissner's membrane.

ANALYTICAL TECHNIQUE.

Because of the limited amounts of fluid available, micromethods had to be developed or adapted specifically for the purpose. The special micropipettes, tubes and general techniques are those previously described for another purpose.[12] The reader is referred to this publication for details of pipetting, mixing, performing colorimetry, etc., on this scale.

The principles of the chloride method and necessary precautions have been published,[12] but the method has been revised to provide increased precision. This is possible because of the greater amount of chloride present. The sodium method is adapted from an earlier procedure.[13] The K measurements were made with two different methods: in endolymph, which has a high K concentration, dipicrylamine[14] was used as a precipitation agent. In the other fluids precipitation was effected with chloroplatinic acid.[15] This latter reagent is more sensitive, but gives less precision at this microscale.

Chloride. To 0.2 μl. volumes of sample or standards (0.1, 0.12 and 0.14 M NaCl) were added 2.5 μl. 0.013 M $AgNO_3$ in 0.5 N HNO_3. After 30 minutes the samples were centrifuged, and a 2.0 μl. aliquot of supernatant fluid was added to 500 μl. of rhodanine reagent. This reagent is freshly prepared by mixing 4 ml. of 1 N H_2SO_4, 1 ml. of 10 per cent gum arabic, and 0.25 ml. of 50 mg. per cent solution of 5-(*p*-dimethylaminobenzylidine)—rhodanine (*p*-dimethylaminobenzalrhodanine, No. 2748, Distillation Products Industries, Rochester, N. Y.). After 30 minutes the samples were read at λ470 mμ. Replicate samples of inner ear fluids had a standard deviation of 0.7 millimoles per liter (0.6 per cent).

Serum was analyzed for chloride by a similar procedure. To 50 μl. of serum or standard (0.090, 0.10 and 0.11 M NaCl) were added 100 μl. 2.2 N HNO_3. After centrifuging, 50 μl. of the supernatant fluid was precipitated with 100 μl. of 0.020 M $AgNO_3$ in 0.1 HNO_3. Color was developed with 5 μl. of the supernatant fluid (obtained by centrifuging) added to 1 ml. of rhodanine reagent. A correction was made for the fact that only about 94 per cent of serum is water and that consequently, when serum is treated with two volumes of nitric acid, the chloride is diluted 2 per cent less than the chloride of standards.

Sodium. To 0.2 μl. volumes of sample, standards (0.025 and 0.150 M NaCl) or blank (water), in 50 mm. pyrex tubes of 2 mm. bore with pointed tips, were added 4 μl. of Barber and Kolthoff reagent,[16] which had been centrifuged before use. The samples were centrifuged after an hour, and a volume of supernatant fluid as close to 4 μl. as possible was removed without disturbing the precipitate. The precipitates were washed ("buzzed") with 4 μl. of a solution consisting of 20 per cent of Barber and Kolthoff reagent and 80 per cent glacial acetic acid Exactly 4 µl of the supernatant fluid was removed after centrifuging (Throughout the precipitation and washing, it was necessary to avoid evaporation by keeping the tubes capped with Parafilm whenever possible) The precipitate was dissolved in 15 µl water, and 10 µl aliquots were added to either 500 µl of color reagent (spinal fluid, perilymph, and 0 15 *M* standard) or to 200 µl of color reagent (endolymph and 025 *M* standard) The color reagent was a fresh mixture of 25 ml. of 50 per cent acetic acid, 0 25 ml of 10 per cent ascorbic acid, and 0 5 ml of 25 per cent potassium ferrocyanide (fresh or frozen)[13] Readings were made after 10 to 60 minutes at λ470 mµ The molar extinction coefficient, is about 12,000 The standard deviation for 0.2 µl samples of perilymph and spinal fluid was 6 millimoles per liter, or 4 per cent This could probably be improved by further study of precipitation and wash conditions.* The precision was, however, sufficient for the present purpose The standard deviation with endolymph was about 1 millimole per liter or 6 per cent.

Serum sodium was determined in a similar manner, even though it might have been measured with a flame photometer, in order to use the same method for all fluids Protein was removed from 50 µl of serum by adding 5 µl of 55 per cent trichloracetic acid and centrifuging Sodium was precipitated from 1 µl of supernatant fluid with 20 µl of Barber and Kolthoff reagent After centrifuging, 20 µl of supernatant fluid were removed and the precipitates were washed with 20 µl. of glacial acetic acid The precipitates were dissolved in 20 µl of water, and color was developed by adding 5 µl of the resultant solution to 1 ml of color reagent. The standard deviation was 2.2 millimoles per liter or 1 5 per cent.

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* A somewhat better procedure used for some of the last samples seemed to be the following 0 2 µl of sample was precipitated with 2 µl of Barber and Kolthoff's reagent. After standing and centrifuging 1 7 µl of supernatant fluid were removed and the precipitate was washed with 2 µl. of glacial acetic acid After centrifuging [illegible] µl of supernatant fluid were removed. Subsequent steps were unchanged

Potassium. To 0.2 μl. volumes of *endolymph* and standards (0.12 and 0.15 *M* KCl) were added 1.7 μl. of 0.03 *M* dipicrylamine in 0.033 *M* NaOH. After standing 30 minutes at room temperature the samples were centrifuged, and a 1 μl. aliquot of the supernatant fluid was added to 1 ml. of 0.005 *N* NaOH and the optical density was measured at λ430 mμ. No precipitation occurred with less than 6 mM K per liter (0.6 mM per liter during precipitation). The molar extinction coefficient is about 26,000 for *increments* of potassium. The standard deviation was 2 mM per liter with standards (1.5 per cent). Duplicate endolymph samples had a standard deviation of 8 mM per liter.

To 0.2 μl. samples of *perilymph* and *spinal fluid* and standards (.004, 0.006, and .008 *M* KCl) and blanks were added 1.6 μl. of 1 per cent $H_2\ PtCl_6\ .6H_2O$ in *n*-butanol. These were centrifuged after one hour at room temperature and 1.6 μl. of supernatant fluid were removed. The precipitates were washed once with 10 μl. of *n*-butanol containing 0.02 per cent chloroplatinic acid and once with 10 μl. of *n*-butanol alone. (The chloroplatinic acid could probably be omitted from the first wash.) The precipitates were dissolved in 40 μl. of 0.1 *N* HCl, and the optical density was measured at λ259 mμ. The molar extinction coefficient was about 12,000 (referred to K).

Protein was measured with 0.6 μl. aliquots by a published colorimetric method.[17]

RESULTS.

A total of 350 analyses were performed on serum, spinal fluid, and labyrinthine fluids from 53 guinea pigs. Only rarely was it possible to make all measurements on fluids from the same animal.

The most striking finding is that Na and K concentrations are almost reversed in utricular endolymph and perilymph (Tables II and III). The endolymph K concentration is 30 times that of perilymph and spinal fluid, whereas the Na concentration is 10 times higher in perilymph than in endolymph.[*] The concentration of the chloride ion is about 10

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[*] It was possible with the flame photometer to confirm the high K concentration in a few larger endolymph samples, although the precision of measurement was less than with the chemical methods. The values found were 109, 137, 144, and 132 millimoles per liter in endolymph, compared to 7 and 5 millimoles per liter in two large samples (1 μl.) of perilymph.

TABLE II.

CHLORIDE, SODIUM AND POTASSIUM OF SERUM, SPINAL FLUID, PERILYMPH AND UTRICULAR ENDOLYMPH.

All values expressed as milliequivalents per liter. The endolymph for one animal had Na and K values of 58 and 76 m.Eq. per liter, without obvious explanation. These data have been omitted from the averages.

	Serum	Serum ultrafiltrate (calculated*)	Spinal fluid	Peri-lymph	Endolymph (utricle)
Chloride	93.9	101	122.4	121.5	107.1
SEM+	1.5		1.0	1.2	1.4
n++	8		19	17	14
Sodium	138.6	138	152.0	150.8	15.8
SEM	1.9		1.8	2.1	1.6
n	14		17	18	12
Potassium			4.2	4.8	144.4
SEM			0.5	0.4	4.0
n			11	13	10
K+Na—Cl		41**	34	34	53

+ Standard error of the mean

++ Number of animals.

* Because the average serum protein was only 4.3 per cent in these young guinea pigs calculation was based on a Donnan ratio of 0.96 and a water content of 965 gm. per liter.

** Assuming 4 m.Eq. K per liter of serum.

TABLE III

INDIVIDUAL ELECTROLYTE VALUES IN SERUM, SPINAL FLUID, PERILYMPH AND UTRICULAR ENDOLYMPH FROM FIVE GUINEA PIGS

All values expressed as milliequivalents per liter

Guinea Pig No	Chloreid				Sodium				Potassium		
	Serum	Sp. Fl.	Peri	Endo	Serum	Sp. Fl.	Peri	Endo	Sp. Fl.	Peri	Endo
48	91	123	120	107	134	156	160	5.6	6.8	6.3	170
49		118	121	106		146	150	19.1	6.7	7.3	143
54		121	122	116		148	144	16.2	3.9	4.4	146
57		121	124	112	149	164	160	16.6		6.0	141
59	94	122	125	111	130	152	154	12.7	2.2	2.7	143

per cent lower in endolymph than in perilymph. No difference was found in the concentration of any of the three ions between perilymph and spinal fluid. It will be noted that the concentration of undetermined anion is substantially greater in endolymph than in the other fluids.

The analyses of *cochlear endolymph* (Table IV) are fewer in number, and the collection of uncontaminated endolymph was much less successful than in the case of the utricle; nevertheless, there are six analyses for Na or K which concur with the utricular data. In an additional case (#101) the data are much as would be expected from a mixture of endolymph and perilymph. Thus it seems quite probable that cochlear endolymph shares with utricular endolymph the unusual Na and K composition.

TABLE IV.

ANALYSIS OF COCHLEAR ENDOLYMPH.

All values are expressed as m.Eq. per liter.

Probably endolymph*			Possibly a mixture of endolymph and perilymph**			Composition not determined***		
Animal	K	Na	Animal	K	Na	Animal	K	Na
No. 101	49	116	No. 104		12	No. 89	109	
No. 106		38	No. 105	10	153	No. 98	175	12
No. 111	150		No. 109	10		No. 107	13	148

* Reissner's membrane was intact. In case No. 101, two holes were drilled into the bony cochlea. The first hole, in the third turn, was too large and the strial exposure considered disadvantageous. Fluid was removed from a second hole in the second turn. Sections revealed disorganization of the stria vascularis in the third turn, with some of the cells disrupted and free in the scala. Leakage from the spiral ligament into the scala media is not excluded.

** Based on observations during the fluid removal or on examination of the sections. Reissner's membrane was intact.

*** Inadequate fixation, distortion, or loss of the celloidin sections.

Protein analyses of labyrinthine fluids are particularly liable to disturbance from contamination with traces of blood. The data (Table V), therefore, set an upper limit to the concentrations of protein present, and the lowest values are perhaps the most reliable. It would appear that all three fluids are very low in protein, that endolymph contains no more protein than spinal fluid, but that perilymph may contain twice as much protein as either of the other fluids.

TABLE V.

PROTEIN CONTENT OF SPINAL AND LABYRINTHINE FLUIDS.

All values expressed as mg. per cent.

Spinal fluid	75,*	24,	24,	13,	21,	28,*	25,	19.			Av.**	21 ± 2
Perilymph	32,	19,	55,	33,	49,	65,	71,	56,	65,	60.	Av.	50 ± 5
Endolymph (utricular)	16,	97,*	11,	14,	26,*	72,*	34,*	152,*	18.		Av.	15 ± 2

* Indicates presence of microscopically visible red blood cells.

** Averages of those not contaminated with red blood cells. The standard error of the mean is indicated.

DISCUSSION.

The present study seems to demonstrate the advantage of the utricle as a source of endolymph, uncontaminated by perilymph. The utricle represents an easily accessible cistern of endolymph. The entire structure can be exposed so that the tip of the pipette is visible at all times. The surface of the utricle can be kept free of fluid for an adequate period of time.

Several of the animals seem to present evidence for the functional importance of the utriculo-endolymphatic valve. This valve, which is an epithelial fold interposed between the utricle and the utricular duct was described by Bast.[18] Observations as to its functional capacity in retaining normal pressure in the utricle and semicircular canals have been made by Bast,[19] Bast and Eyster,[20] and Perlman and Lindsay,[21] The sections from three animals showed that the vestibular cochlear duct had been opened during the surgery; yet, the utricles showed normal distension. Judging from the chemical analyses, no mixing of perilymph and endolymph had occurred. These findings appear to present additional evidence for the ability of the utriculo-endolymphatic valve to close off the utricle and semicircular canals from the rest of the membranous labyrinth when normal pressure relations are changed.

The chemical findings agree with those of Kaieda,[1] for the shark, in regard to the similarity of spinal fluid and perilymph, but disagree in regard to the composition of endolymph (Table I). It is possible that the labyrinthine fluids of the fish might represent an adaptation to environmental and anatomical differences. The somewhat lower chloride found in endolymph is not in agreement with the data of Ledoux[9] for the cat (Table I).

The data presented show that endolymph, at least in the guinea pig, is unique among known extracellular fluids and resembles intracellular fluids in regard to Na and K content. The significance of this unusual electrolyte pattern is not obvious. It is difficult to reconcile the high K concentration of endolymph with the positive DC potential of the scala media demonstrated by Bekesey.[22] The situation is just the reverse of that found for muscle and nerve, in which a negative potential is associated with the side of the membrane having a high K concentration. Any attempt to resolve this difficulty must take into consideration the structure of the scala media. The stria vascularis at its lateral wall and the complex sensory structure inferiorly may well add to the complexity of the processes occurring inside. It is possible that the DC potential and the K/Na ratio are related in some other way than in nerve and muscle fiber.

If the prevalent concept of the basilar membrane as one boundary of the cochlear duct is accepted and the tunnel is supposed to be filled with endolymph, then it would seem that the high K content should seriously interfere with the conduction of the nerve fibers that cross the tunnel. Davis, Tasaki and Eldredge[23] believe that their experiments with the injection of KCl into the scalae and their measurements with an exploring electrode show that the tympanic boundary of the cochlear duct should be placed at the reticular lamina covering the organ of Corti. The nerve, therefore, would be separated from the endolymph. The fact that the nerves do conduct impulses in spite of the high K content of endolymph clearly supports this concept.

SUMMARY.

Perilymph, cerebrospinal fluid, and endolymph from the utricle of the guinea pig were analyzed for Na, K, Cl, and protein. Cochlear endolymph was analyzed for Na and K, and blood serum was analyzed for Na and Cl. Special microchemical methods were devised for the purposes, since it was necessary to work with samples of the order of 0.2 μl. (.0002 ml.). Endolymph was found to have a K concentration 30 times that of perilymph, and a Na concentration only a tenth as high as perilymph or cerebrospinal fluid. The Cl of endolymph was found to be 90 per cent of that in perilymph and cerebrospinal fluid. Perilymph and cerebrospinal fluid were indistinguishable in K, Na, and Cl content. All three fluids are low in protein, but perilymph may contain twice as much as either of the other fluids.

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LARYNGEAL SPASM.*†

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INTRODUCTION AND DISCUSSION.

This is a report of work undertaken to secure, if possible, direct evidence on the nature of laryngeal spasm. We proceeded on the basis that laryngeal spasm depends upon the operation of the usual mechanism for protection of the trachea and lungs by glottic closure. This involves an understanding of the anatomy and physiology of the larynx. The following scheme was used as a guide:

I. Structures involved: A. Nervous—
  1. Afferent pathways—
    a. From the surface of the pharynx and the larynx *i.e.* Cranial nerves, V, IX, and X.
    b. From any other structure (in theory at least any afferent nerve may be involved).
  2. Central apparatus (*i.e.* the entire central nervous system) connecting the pathways noted above with the
  3. *Efferent* pathways in cranial nerve X (vagus).
    a. The superior laryngeal nerve (external branch to the cricothyroid muscle).
    b. The recurrent laryngeal nerve (branches to all other laryngeal muscles).

B. Muscular—
The various muscles, abductor and adductor of the glottis. These are *striated* (skeletal or voluntary) muscles.

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Although the Xth cranial nerve (vagus) is the important connector structure, it is well to note that neither the innervation nor the effectors (muscles) are autonomic.

The peripheral nerves are *medullated;* there are no *peripheral efferent ganglia;* and the muscles are *striated.*[3,4,5] There is, therefore, no reason to expect that autonomic blocking agents (*e.g.* atrophine) will have any direct effect on the structures involved.

II. Physiology—A discussion of the innervation of the larynx with bibliographies is given by Cole[1] and by Murtagh.[2] The largest *adductor* of the glottis is the cricothyroid muscle which is supplied by the external branch of the superior laryngeal nerve. The important *abductor* is the posterior cricoarytenoid muscle supplied by the recurrent nerve.

In brief, laryngeal spasm may be orginated

1. *Reflexly*—
    - a. By intensive stimulation of pharyngeal or laryngeal receptors
    - b. By intense (painful?) stimulation of any receptor, (6) (7);
2. *Centrally* by altering the state of the central apparatus (*e.g.* strychnine poisoning); and
3. *Peripherally*—
    - a. By stimulating an efferent nerve, or
    - b. By stimulating the muscles directly.

Laryngeal spasm may be prevented or stopped by avoiding the circumstances listed above. This may be accomplished in

1a. By local anesthesia of the pharynx and the glottis or more immediately by not allowing secretions to accumulate; *1b.* and *2.* By adequate general anesthesia; and *3.* By avoiding trauma in the neck region.

The effects of *1*, *2*, and *3a* may, of course, be prevented by the use of a neuromyal blocking agent (*e.g.* curare).

Proper use of an intratrachial tube obviously avoids most difficulties.

METHOD.

Goats were used as experimental animals. The basic anesthesia was pentobarbital sodium (Nembutal) given intraperitoneally (0.45 Gm/kilo).

A tracheal cannula was inserted well down in the neck to leave the upper trachea and larynx free for experimental procedures. The recurrent nerve was visualized on each side, and the esophagus was tied to prevent regurgitation. The pharynx, in most experiments was packed with gauze in an attempt to keep secretions from entering the glottis.

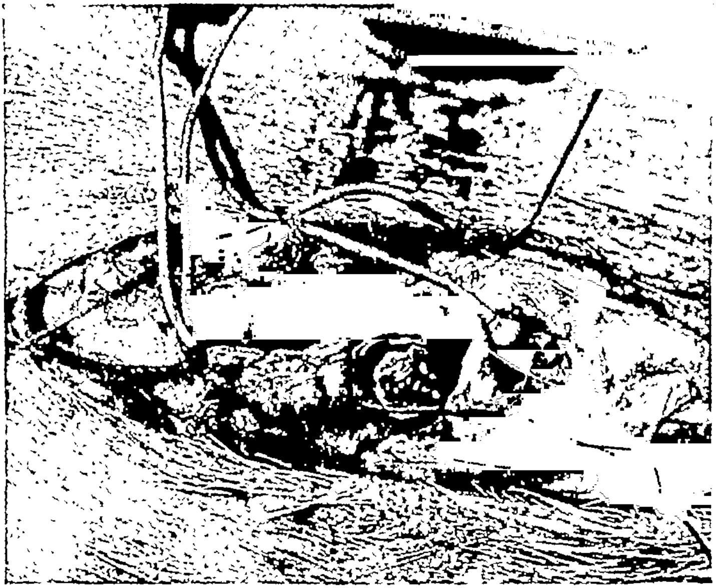

Fig. 1. A Photograph showing the Placement of the Needle Electrodes.

The trachea was opened between the second and third rings, taking care to avoid the recurrent nerve. The larynx was elevated and the dorsal portion of the cricoid cartilage freed to expose the posterior cricoarytenoid muscles bilaterally.

Electrodes were inserted in each *posticus* muscle (an abductor); the larynx was laid back in place; and the lead wires from the electrodes were anchored by a suture around the third tracheal ring. Similar electrodes are placed in each cricothyroid muscle (an adductor) (see Fig. 1).

The electrodes are made by soldering insulated, flexible lead wires to the hubs of 31-gauge intradermal needles. The needles (hubs included) are then dipped in insulating varnish (Tygon). When the varnish has hardened a length of 1 to 1.5 mm. at the point is bared by dipping in solvent. The needles are inspected under a microscope and are tested by passing direct current electricity at 500 volts between the needle and a strip of metal (anode), with both immersed in a dilute saline solution. If bubbles form on the needles anywhere except on the bared tip the electrode is recoated.

A large indifferent electrode is fastened to shaved skin between the rami of the lower jaw, using collodion. A ground electrode is placed in a similar manner on the vertex.

The action potentials of the individual muscles are recorded against the potential of the indifferent electrode by leading to an eight channel Grass Model III encephalograph. Any potential change which is recorded by all channels simultaneously will have its origin under or near the indifferent electrode, and will probably represent a swallowing motion.

The placement of the individual electrodes is facilitated by temporarily connecting each electrode to a separate preamplifier, which is in turn connected with both a cathode ray oscilloscope and an audio amplifier—loud speaker set-up. While viewing the pattern on the scope and listening to the sounds from the speaker, the electrode is manipulated until it is near an active motor unit.

The recorded electrical response is principally from the units in the neighborhood of the electrode. Repeated experiments, however, should give a reasonable picture of the response of the muscle as a whole.

An accordion type pneumograph is placed around the chest, connected with a strain gauge bridge, so arranged that the circumference of the chest is represented by an envelope described by one of the pens of the encephalograph.

A microphone connected with one channel of the encephalograph is placed over the tracheal cannula to indicate the instant and, in a general way, the force of the expiratory blast.

EXPERIMENTAL RESULTS.

Controls and Local Reflexes—

Until we had considerable experience with the preparation; had learned to keep the glottis scrupulously clean, and had arranged to deliver the pentobarbital sodium continuously through an intravenous cannula, we secured no records from the muscles which were not indicative of continuing contraction (spasm). Records obtained from a preparation showing what we consider to be a normal respiratory pattern are shown in Fig. 2. Here the muscle action potentials are shown during quiet respiration. Throughout expiration the cricothyroids (adductors) show moderate activity. The greatest activity of the posticus muscles (abductors) is late in inspiration. Synchronous with the activity of the posticus group there is also some slight discharge from the cricothyroids. Both muscle groups show periods of relative inactivity.

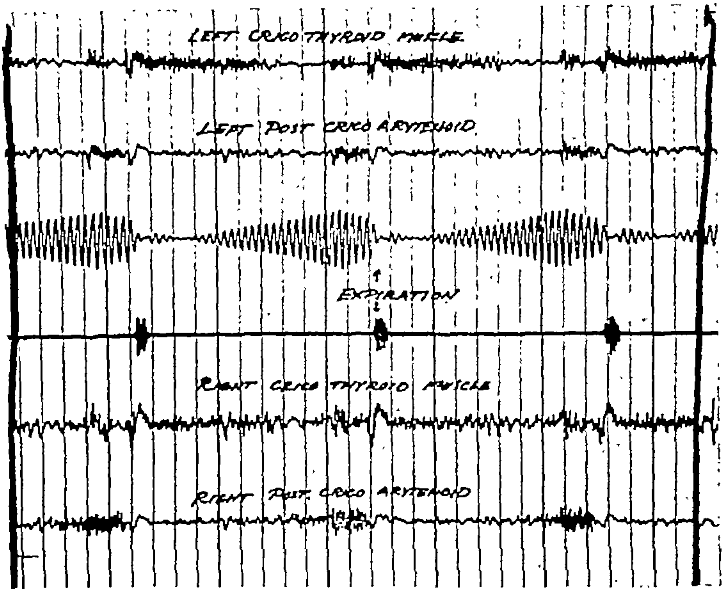

Fig. 2. Experiment of July 9, 1953—Goat. Channels 1, 2, 5 and 6 Electromyograms. Channel 3 envelope of chest size, Channel 4 microphone showing Expiratory blast. The time interval between vertical lines is 0.4 sec.

The electrical activity, when spasm is present, is shown in Fig. 3. Here there is no evidence of respiratory rhythm in the action potential records. The potentials are increased, and the firing continues undiminished throughout the respiratory cycle. This record was taken just before alcohol was applied to the right recurrent nerve. Fig. 4 shows the cessation of activity in the right posticus five minutes after the application of alcohol to the nerve. As a further control the left recurrent nerve was cut. The results are shown in Fig. 5. There are no action potentials from the right posticus muscle (previous alcohol block), and the action potentials from the left posticus cease on section of the nerve. There is a flurry of activity in the left cricothyroid at the moment of section, followed by reduced activity of both cricothyroids for a period of 0.10-0.12 seconds. We are at present uncertain of the explanation of this transient inhibition.

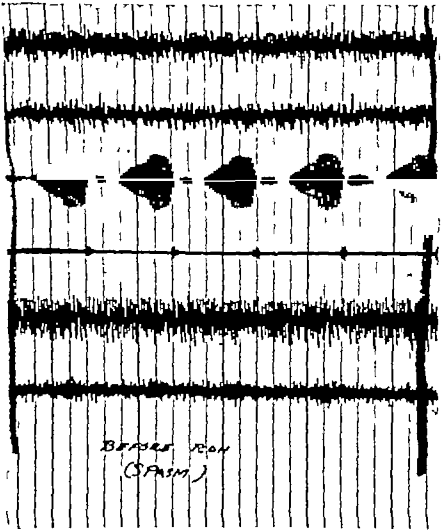

Fig 3 Experiment of July 9, 1953—Goat. Channels as in Fig. 1. Time interval between vertical lines is 1.0 sec.

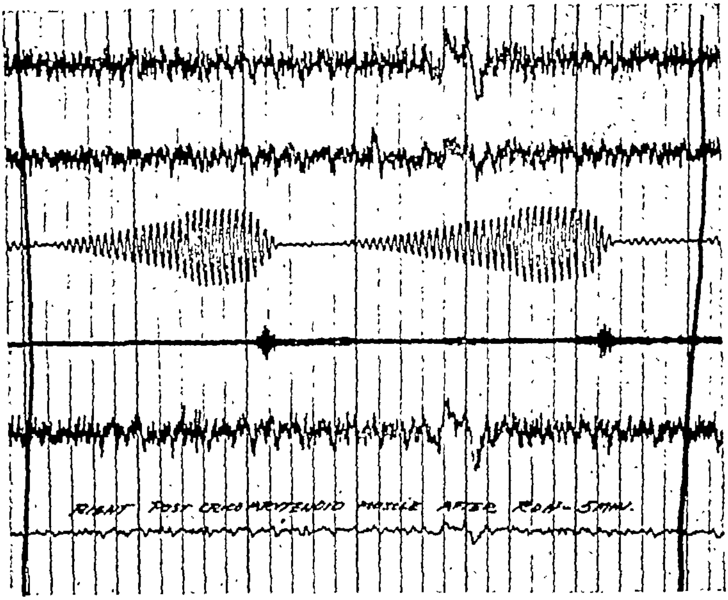

Fig. 4. Experiment of July 9, 1953 — Goat. Channels as in Figs. 2 and 3. Time 0.4 sec. between lines.

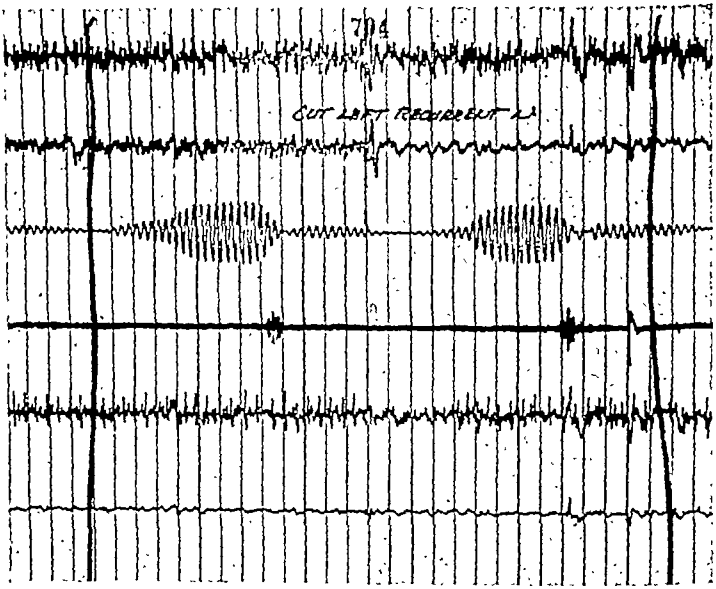

Fig. 5. Experiment of July 9, 1953 — Goat. Details as in Fig. 4.

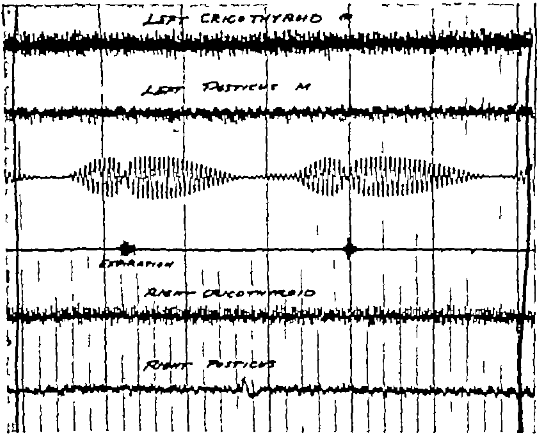

Fig 6 Experiment of July 6 1953—Goat. Channels as in Fig 2 Time 0.4 sec. between lines.

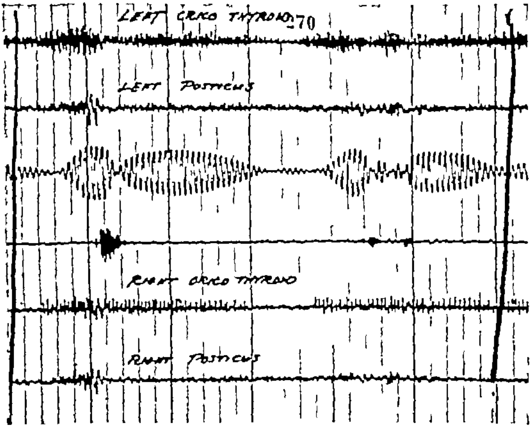

Fig 7 Experiment of July 6 1953—Goat Channels and Time as in Fig 6.

The effect of foreign material in the glottis is shown in Fig. 6. The electrical records indicate continuing contraction of all four muscles. The respiratory record is marred by the fact that the pneumograph apparatus is not in balance, and that both inspiratory and expiratory movements appear as apparent increases in circumference of the chest. The microphone record, however, gives a correct indication. After the larynx had been freed of blood clots and saliva, washed with saline and sucked dry, the character of the responses changed to the pattern shown in Fig. 7. Here rhythmic respiratory changes in action potentials replace the spasm shown in the previous figure.

The effects of cutting the internal (sensory) division of the superior laryngeal nerve is shown in Fig. 8. Here there is a transient inhibition of the activity of the muscles, particularly of the cricothyroids. Recovery is nearly complete at the end of a minute (see Fig. 9). Twenty seconds later spasm had returned and the record resembled Fig. 8.

EFFECTS OF ANESTHETIC AGENTS.

Pentobarbital Sodium (Nembutal)—

Pentobarbital sodium was used as the usual agent, both for induction and maintenance. With a good preparation and adequate anesthesia, records similar to those shown in Fig. 2 were obtained. To note the effects of other agents the continuous, dilute, intravenous pentobarbital was disconnected, and the other material injected through the polyethylene tube in the femoral vein.

To note the effect of a large dose of pentobarbital, the animal was allowed to become light and the drug was injected between the arrows as shown in Fig. 10. Because of improper switching the action potentials are recorded from the left and right cricothyroid muscles only. The remaining two channels are also left cricothyroid. Chest size and the expiratory blast are shown in channels 5 and 6, the slower swings (synchronous in all electromyograms) represent swallowing. Within eight seconds all activity in the muscle records ceased and ventilation had stopped. Artificial respiration was given for thirteen minutes, when spontaneous respiration again began.

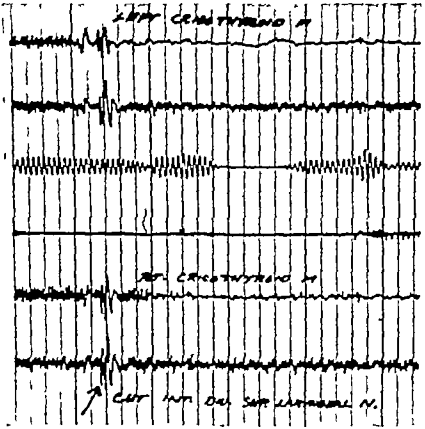

Fig 8 Experiment of July 6 1953—Goat. Channels as in previous figures. Time 0.4 sec between vertical lines.

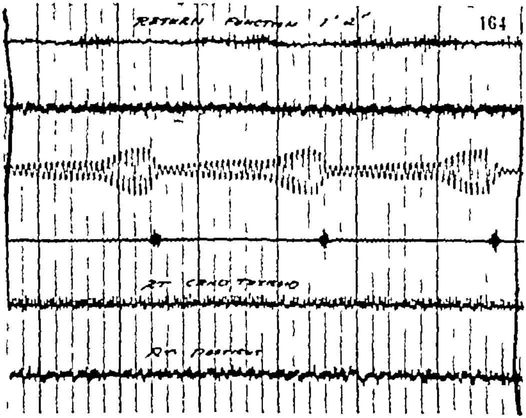

Fig. 9 Experiment of July 6 1953—Goat. Channels and Time as in Fig. 8

During this time, as observed on the oscilloscope and loud speaker there were no action potentials from either cricothyroid, and only a few small disturbances from either posticus

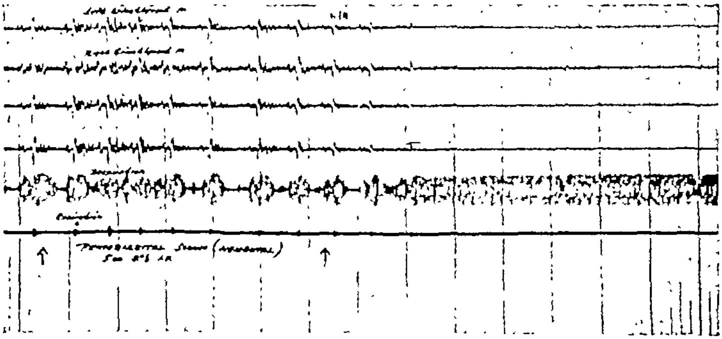

Fig. 10. Experiment of December 3, 1953 — Goat. Time 1 sec. between vertical lines. See text for description.

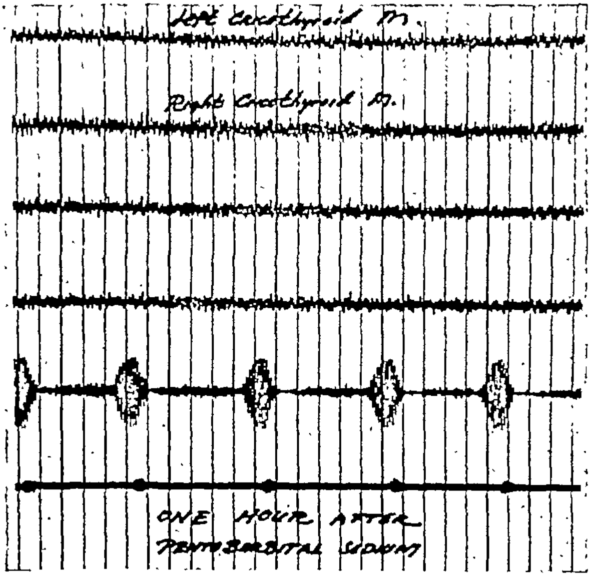

Fig. 11. Experiment of December 3, 1953 — Goat. Details as in Fig. 10.

At the end of eighteen minutes, although respiration was continuing, there were no evidences of activity of the glottic musculature. One-half hour after the injection there were a few rhythmic bursts of muscle potentials, and after an additional five minutes (no anesthesia during this time) continuous firing (spasm) was resumed. Figure 11 shows conditions at the end of an hour. *After some thirty-five minutes the slow* intravenous pentobarbital had been resumed. These results are typical of our experience with pentobarbital.

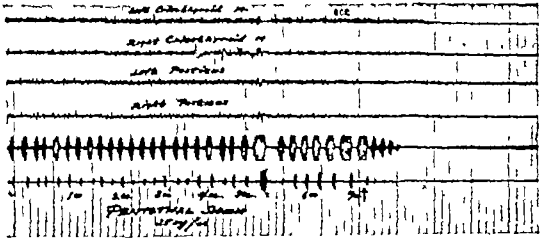

Fig 12 Experiment of December 10, 1953—Goat Myograms as labelled with Respiration and Expiratory Blast in Channels 5 and 6 Rate of injection shown on record.

Pentothal Sodium—

This agent is reputed to cause laryngospasm in some experimental animals[7] and has a bad name in this respect among anesthesiologists. We made some half dozen experiments, using it, and will report on one run only. In Fig. 12 is a record of an injection, and some twenty seconds after the administration. The animal, deliberately light, showed electrical evidences of continuing activity, upon which was superimposed a respiratory rhythm. The record is sufficiently graphic and shows that ventilation stopped within five seconds of the final cc. of pentothal, and that all large potentials had disappeared within ten seconds. Inspection showed the cords motionless. Artificial respiration was carried on for about four minutes. Respiratory rhythms began in the myograms at that time, and ventilation resumed in about a minute. The conditions at the end of eight minutes showing a slight reduction in electrical activity, particularly in the abductors, is shown in Fig. 13. In none of the experiments was there any evidence of increased electrical activity of the muscles.

No other anesthetic agents were tried.

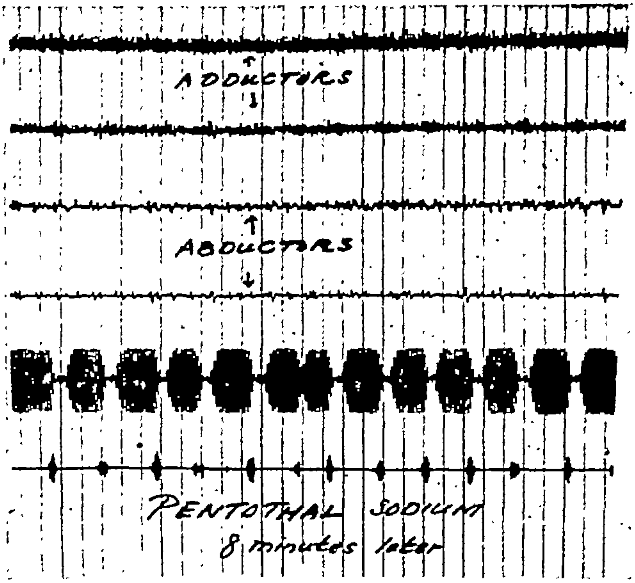

Fig. 13. Experiment of December 10, 1953 — Goat. Channels as in Fig. 12. Time 0.4 sec. between lines.

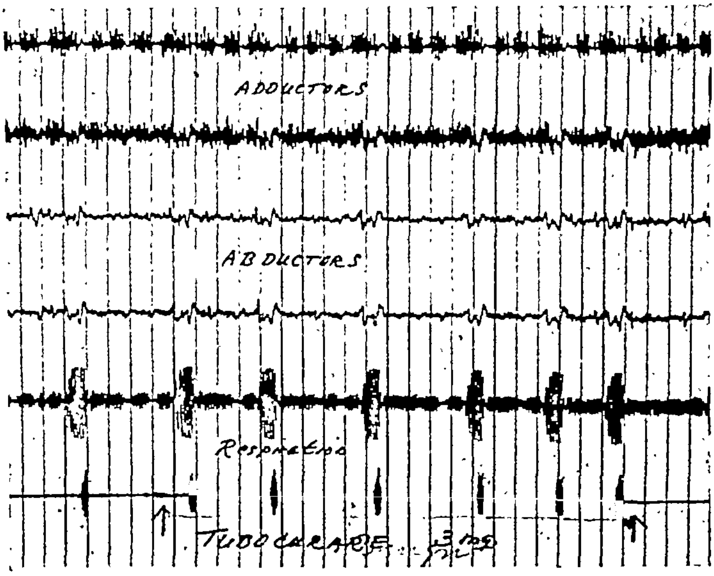

Fig. 14. Experiment of December 10, 1953 — Goat. Details as in Fig. 13.

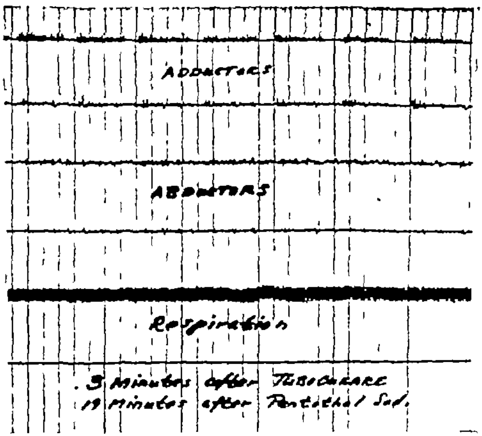

Fig 15 Experiment of December 10, 1953 — Goat. Details as in Fig 13

MYONEURAL BLOCKING AGENTS.

Curare—

In several experiments curare (tubocurarine chloride) was injected in various amounts and at various rates. In every case respirations stopped, and the action potentials from the glottic muscles diminished, and then disappeared. The records of Figs. 14 and 15 were chosen because they represent a continuation of the records shown in Figs. 12 and 13. The curare was given sixteen minutes after the pentothal. In just over one minute the respirations had stopped, and the muscle potentials were reduced, although they still showed respiratory rhythms. At the end of three minutes the changes are shown (see Fig. 15). Artificial respiration was not given. The electromyograms vanished in six minutes, although swallowing potentials continued for several more minutes.

Succinylcholine—

Succinylcholine chloride (Anectine) was given on several occasions. Although its mode of action is considered to be quite different from that of curare, the results are very similar. Respiration was depressed before any changes were noted in the electrical records from the glottis. In some experiments, particularly when using larger doses, there was evidence of a transient augmentation of potentials (in one case a slight, general convulsion) before the depression set in. From the records on hand, one set is chosen because it illustrates a number of points. A moderate dose (5 mgms. diluted 1 to 5) was slowly injected but not washed in. Within one minute respiration had stopped, though the action potentials of the glottic muscles continued. Artificial respiration was given for four minutes when shallow ventilation returned. Evidences of increased glottic activity appeared, and the condition at the end of ten minutes is shown in Fig. 16. At this time the cords were abducted. The intravenous pentobarbital, which had been stopped during this time, was then started. The succinylcholine remaining in the tubing was in this way washed into the circulation.

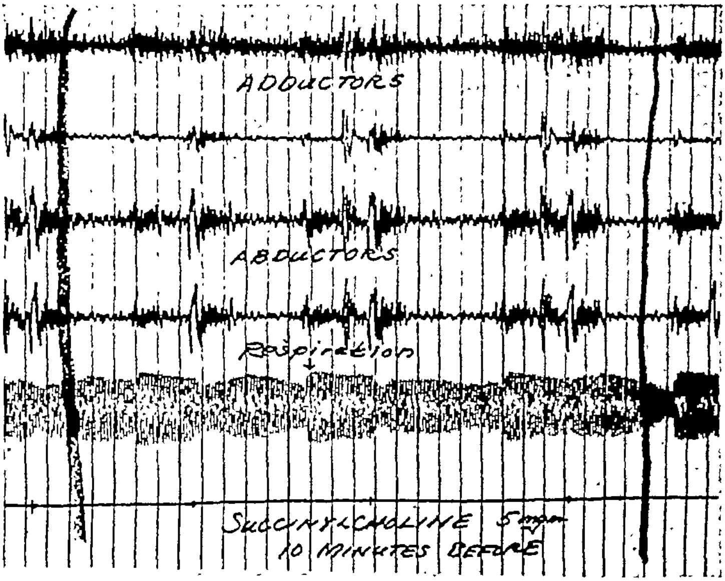

Fig. 16. Experiment of December 17, 1953 — Goat. Details as in Fig. 13.

We have no way of knowing how the dose was divided. The electrical changes in the glottis continued as respiration was

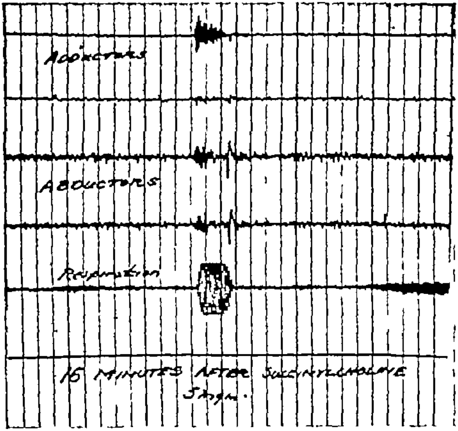

Fig 17 As in Fig 16

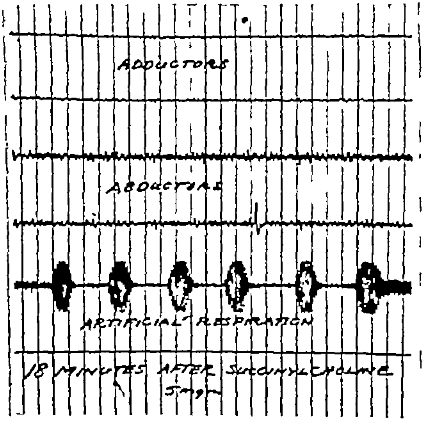

Fig 18 As in Fig 16

again depressed. Six minutes later the record of Fig. 17 was obtained. Respiration is very slow, the expiratory blast is negligible, but the action potentials are certainly not diminished. Figure 18 shows the record at the end of eighteen minutes. Artificial respiration was required for an additional five minutes, and then the normal pattern returned.

No other blocking agents were tried.

CONCLUSIONS.

Using the electrical changes which take place in striated muscles as evidence of activity, and considering that the size and the frequency of these potentials are indications of the quantity of activity taking place, we have studied the behavior of the glottis under various circumstances. Because of technical difficulties only two pairs of muscles (one adductor and abductor) were investigated.

With the data at hand it has been shown that experimental laryngospasm may be produced reflexly by stimulation of the laryngeal mucosa (or any sensory nerve leading from the larynx).

The usual accumulation of secretions is adequate to set up this spasm, if the depth of anesthesia is inadequate.

No evidence was obtained to show that the fixed dose anesthetics, pentobarbital and pentothal, increase the activity of the laryngeal musculature. All evidence was that these materials given in adequate amounts depress (centrally) the responses of these muscles. This probably holds true for all central nervous system depressants.

Graphic evidence of the action of the neuromyal blocking agents (curare and succinylcholine) has shown the effectiveness of these agents, and has strikingly illustrated the respiratory depression that follows their use. The glottic musculature fails after respiratory paralysis has set in and before swallowing movements have stopped.

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THE INCREASING INDICATIONS FOR TRACHEOTOMY.*†

G. S. Fitz-Hugh, M.D.,
and
W. C. Morgan, Jr., M.D.*
(By Invitation)
Charlottesville, Va.

In 1940, one of us (G.S.F.) presented a study of 100 patients[1] upon whom 102 tracheotomies had been performed. The primary motive for this survey was to learn something of the risks and complications resulting from such a procedure. The results indicated that the risks involved in this surgical procedure were negligible, and that the only complication occurring with any degree of frequency was pneumothorax; however, the pathologic condition for which tracheotomy was necessary was responsible for a rather high incidence of death in this group.

Again after a period of some fourteen years the same type of study has been made, with some difference in that the primary aim in this later group was to examine the indications for tracheotomy. It is apparent to all practicing medicine and surgery now, that these indications have increased in scope and number in the last few years. Perhaps it is better stated, that these reasons have always existed, but that the recognition of such recently, has come with a better understanding of the physiology of respiration as applicable to clinical, rather than laboratory medicine. The main impetus for the development of this additional knowledge results from the opportunity to study the respiratory problem afforded by the many unfortunate poliomyelitis victims. Credit must be given to Wilson,[2] Galloway,[3] Cummings,[4] Priest[5] and Bower,[6] their co-workers and others, for their invaluable contributions to this subject. Harris[7] in 1952 published the

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results of his efforts to investigate the clinical value of tracheotomy in the prevention and management of the various types of pulmonary difficulties encountered in poliomyelitis. His conclusions were that a reduction in poliomyelitis fatalities could be accomplished by tracheotomy in certain cases, particularly when performed early. This has been the experience of many other otolaryngologists.

The need for tracheotomy for obstructive conditions in the hypopharynx and larynx, due to edema of the mucous membranes, as the result of inflammation, allergies, trauma and chemicals, is well established and recognized. Intrinsic mechanical obstruction, due to vocal cord paralysis, foreign bodies and tumors is commonplace; extrinsic pressure from cervical masses, neoplastic or inflammatory in nature, causing embarrassment in respiration, is not unusual.

As mentioned, there has been an increase in the indications for tracheotomy in recent years. We have noticed this in our experience, as will be presented later, and we have also observed this trend recorded in the literature.[8,9] Specifically, Carter and Giuseffi[10] have reported the use of tracheotomy in chest injuries; Atkins,[11] in the prevention of pulmonary complications in postoperative and debilitated patients; Lahey and Hoover,[12] following thyroidectomy; Echols[13] and McCart[14] and their co-workers in the management of head injuries; Herzon,[15] Creech[16] and their collaborators in the treatment of tetanus; Taylor and Austin[17] in the handling of pulmonary complications in neurosurgical patients; Collins, et al.,[18] in eclampsia; Sloan,[19] in leprosy; Bofinkamp,[20] in botulism; Lewy[21] in barbiturate poisoning; Everett[22] in laryngotracheobronchitis, and Colvin and Morrison[23] in acutely ill patients.

The basic reason for tracheotomy is to permit a normal exchange of air in the alveoli of the lung for the absorption of oxygen and the elimination of carbon dioxide (Harris[7]). Any significant interference with this mechanism will result in rapid death, or if corrected just prior to this stage, very possibly in irreversible nerve tissue injury, with death secondary to complications therefrom. If the patient survives, permanent embarrassment and compromise in the efficient function of the human mechanism may ensue. Anoxia, hypoxia, carbon dioxide retention in the blood and acidosis are not tolerated for long periods. In this regard, it has become apparent that obstructive conditions, such as retained tracheobronchial secretions, may well lead to subclinical anoxia and hypercapnia with cerebral manifestation, which in turn may be confused with, or be blamed upon, the primary agent, such as infection (encephalitis) or hemorrhage (trauma). The correction of the responsible medium producing the anoxia by establishment of an adequate airway may cause a marked difference in the future well-being of the patient.

A total of 150 consecutive patients upon whom tracheotomies had been performed, were studied. Two patients in the group required two procedures; the second one in each case was performed prior to laryngeal surgery, which had been unsuccessful on the first occasion. Thus a total of 152 tracheotomies were available for consideration; however, the two extra procedures were not considered specifically in our statistics, as such detail seemed irrelevant. The statistics upon 150 tracheotomies are presented.

In our survey we have divided the indications for tracheotomy into three known groups, with a possible fourth group as a questionable and poorly understood one (see Table I):

TABLE I.

INDICATIONS FOR TRACHEOTOMY.

1. Fixed Obstruction to Upper Airway.
   (Rapid Obvious Anoxia)
2. Fluid Obstruction to Lower Airway.
   (Slow Obscure Anoxia)
3. Prophylactic.
   (To prevent 1 and/or 2)

Total Number of Patients Having Tracheotomies........150

1.	80	53.0%
2.	30	20.0%
3.	40	27.0%

First were those that exhibited the manifestations of an impaired airway with anoxia due to more or less fixed mechanical obstructions, such as encountered in a neoplasm of the larynx or bilateral cord paralysis. The degree of impairment varied, of course, but in all cases tracheotomy was indicated. There is little room for confusion in this group.

The *second group* included those cases in which the airway was compromised by a fluid mechanical obstruction, such as an excessive accumulation of material in the tracheobronchial tree from repeated aspiration of oral fluids, hyperfunction of the secretory elements of the tracheobronchial membranes, and/or failure in the cough mechanism.

The *third group* was composed of those cases in which it was anticipated that the airway would eventually be inadequate because of the development of one or both of the conditions exemplified in the first two groups, as for example, in surgery of the upper respiratory or digestive tract such as an arytenoidectomy or extensive cervical dissections. Tracheotomy was performed as a prophylatic measure.

Spasm of the larynx superimposed upon some of the conditions previously mentioned and augmenting respiratory distress undoubtedly hastens the necessity for tracheotomy. We doubt seriously that spasm of the larynx *per se* will cause sufficient trouble to be classified as an indication for tracheotomy, although we certainly do not feel secure in this opinion at the present time.

As one would anticipate, in a general hospital with an active otolaryngologic service, the larger group of tracheotomies was in the static obstructive, or first category (see Table II). Eighty patients were so classified. The obstruction in 23 patients was due to carcinoma of the larynx, or of some structure above the larynx. In 22 incidents, the obstruction was the result of inflammatory conditions such as laryngotracheitis, diphtheria, Ludwig's angina, and one unusual case of pemphigus. As the result of surgery upon the thyroid gland (non-malignant), 11 patients required tracheotomy because of subsequent obstruction to the airway.

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Nine children who were admitted with pulmonary foreign bodies, needed additional airway afforded by tracheotomy because of the nature of, and the reaction to, the foreign body, plus the edema incurred as the result of instrumentation in its removal. Trauma to the airway from external forces, such as sustained in automobile accidents, was responsible for the procedure in four incidents. Tracheotomies were performed upon four patients with bilateral vocal cord paralysis, the causes of which were obscure. Two children with papillomata of the larynx needed the augmented airway afforded by tracheotomy.

TABLE II.

INDICATIONS FOR TRACHEOTOMY.

1. Fixed Obstruction to Upper Airway. Conditions:	Number of Patients
Carcinoma	23
Inflammatory	22
Thyroid Gland Surgery	11
Pulmonary Foreign Bodies	9
Trauma	4
Bilateral Cord Paralysis	4
Papillomata, Larynx	2
Miscellaneous:	
Stenosis (scar), Larynx	1
Gumma, Larynx	1
Ulceration (Chemical), Larynx	1
Pyogen. Granuloma, Larynx	1
Edema (Endotr. Tube), Larynx	1
	80

Single cases requiring tracheotomy were: stenosis of the larynx, due to scarring as the result of X-ray irradiation and chondritis, gumma of the larynx, pyogenic granuloma of the larynx, ulceration secondary to the ingestion of bichloride of mercury, and laryngeal edema secondary to prolonged endotracheal intubation used as an adjunct in the administration of an anesthesia.

In the second group of cases (see Table III), tracheotomies were performed in order to facilitate the cleansing of the fluid collection in the tracheobronchial tree and thus prevent anoxia from this source. Thirty patients were placed in this category, somewhat less than had been anticipated. Neurological conditions, such as spinal cord and brain lesions, were responsible in seven cases. Trauma to the lower face, neck and chest necessitated tracheotomies in eight patients. A medical group composed of diseases such as poliomyelitis, meningitis, pneumonia, uremia; and tetanus were responsible for 13 procedures. The remaining two cases were: one of amyloidosis of the trachea and one of oral neoplasm.

TABLE III.
INDICATIONS FOR TRACHEOTOMY.

2. Fluid Obstruction to Lower Airway. Conditions:		Number of Patients
Neurological Group		
Brain Tumors	3	
Amyotrophic lat. Sclerosis	1	
Postop. Lobotomy	1	
Guillain-Barre Synd.	1	
Cardiac Arrest	1	7
Medical Group		
Poliomyelitis	6	
Uremia	3	
Meningitis	2	
Tetanus	1	
Pneumonia	1	13
Traumatic Group		
Face, Neck, Chest	8	8
Miscellaneous Group		
Neoplasm of Mouth	1	
Amyloidosis, Trachea	1	2
TOTAL		30

TABLE IV.
INDICATIONS FOR TRACHEOTOMY.

3. Prophylactic Conditions:	Number of Patients
Extensive Head and Neck Surgery	35
Traumatic Surgery	4
To Facilitate Anesthesia	1
TOTAL	40

There were 40 cases in the third classification (see Table IV). It is in this group that tracheotomy was performed prior to the development of conditions for which tracheotomy was performed in Groups I and II. In other words, this was the group in which prophylactic tracheotomy was in order. Practically all of the patients in this category were those requiring extensive surgery upon the upper respiratory and digestive tracts, such as the tongue, jaws, etc., in an effort to eradicate carcinoma. In a few this was performed at the time of repairing injuries secondary to trauma. One particular case in this class is worth mentioning, in that in our experience it was the first time such an indication had occurred: An underdeveloped and ill 9-months-old child was tracheotomized prior to chest surgery in order to facilitate the administration of a general anesthetic.

From the above-mentioned cases, we see a more liberal use of tracheotomy with an increase in the number of indications, as exemplified in Groups II and III. We are not certain but that these conditions have been considered as indications all along, to a certain degree, and that the increase is relative, in that the added publicity has focused our attention upon this method of treatment. For instance, in our previous study, a case of poliomyelitis in 1933 received a tracheotomy in order to facilitate the cleansing of the tracheobronchial tree.

With the increase of poliomyelitis, there has been an increase in the number of tracheotomies in the treatment of this disease. Also, since 1933, there has been an actual decrease in conditions causing upper respiratory tract obstruction such as diphtheria, laryngotracheitis and foreign bodies. With the development of improved surgical techniques and antibiotics, more extensive surgical procedures are being carried out, and this is reflected in the enlarged number of prophylactic tracheotomies (third group).

In this recent study special care was made in an attempt to analyze the risk and complications of tracheotomies. This was done because some physicians consider the procedure a hazardous one. Apparently this is not true, for in not one case could we find that the procedure had prolonged the illness of the patient or had hastened death. Obviously, in the vast majority of cases, the opposite occurred. The only complications of note, occurring in this series as the result of tracheotomy *per se,* were mediastinal emphysema and pneumothorax.

Pneumothorax, recognized by radiological and clinical examination, was present in only three recorded cases. We believe that this figure is incorrect, because if routine chest X-ray examinations had been made following tracheotomy, it is likely that more cases of pneumothorax would have been disclosed. The three cases mentioned as recorded complications recovered promptly. Subcutaneous and mediastinal emphysema was often present, but not to a degree to cause concern.

It was recorded in one case that expired within 12 hours after tracheotomy, that more than the usual amount of blood was noted from the wound postoperatively, but that after careful consideration, it was the opinion of the attending staff that this excess blood loss was not a factor in the cause of death. The expected slight wound infection and tracheobronchitis, secondary to the mechanical trauma resulting from the cannula and aspirating tubes, were not considered as complications, as they never reached any degree significant enough to influence the clinical course of the patient.

There were no cases of mediastinitis, tracheal granuloma, or apnea following relief of the obstruction. Eight patients expired within a period of 12 hours after tracheotomy, death in each instance being due to the primary disease or condition requiring the operation. No difficulty was encountered in decannulating any patient in whom this was desired. Davis[24] and his group report in their recent study a higher percentage of complications, but theirs was made from a more critical viewpoint than ours.

As in our previous survey[1], an effort was made again to classify the cases according to the degree of respiratory difficulty requiring tracheotomy. We considered elective cases as those in which respiratory difficulty was not present, tracheotomy being performed in anticipation of such a status, or to facilitate future or present therapy. Emergency cases were those in which total asphyxia was imminent, and the procedure was carried out with little regard to sterile technique or to the assembly of any more of a set-up than that supplied by the emergency tracheotomy tray.

Between these two extremes was classified our so-called intermediate group, in which definite respiratory difficulty or anoxia was present, but to such a degree that the procedure could be planned and performed with the maximum degree of orderliness, safety, and comfort to the patient and surgeon. In the total survey of 150 cases, 72 were elective procedures, 51 were in the intermediate group, and 27 were classified as emergencies, four of the latter having been performed on the wards, or in the emergency room without benefit of the emergency tracheotomy trays.

Comment: A majority of the tracheotomies were carried out by otolaryngologists, but an increasing number were performed by the thoracic and general surgeons. Evidence was present that those performing the tracheotomies other than the otolaryngologists in some instances, were in need of more knowledge of the procedure to obtain the maximum benefit therefrom. This was particularly true in the after-care of the patient.

It is evident also that the indications for tracheotomy, as well as some basic principals of the technique and after-care, should be given more consideration in undergraduate teaching. Tracheotomy is occasionally the solution to one of the few true emergencies in the practice of medicine, respiratory distress, and every physician should be prepared to aid in the solution of the problem.

SUMMARY.

A survey was made of 150 consecutive patients who had undergone tracheotomy. In comparing this study with one made some years previously, it was evident that the indications for tracheotomy had increased, and it was also apparent that this increase was due, in the main, to a better understanding of the clinical physiology of respiration. The fact which had not been universally recognized previously, was that obstruction in the lower respiratory tract from a collection of fluid, could be responsible for anoxia and hypercapnia, as well as, but more insidiously than the well-recognized upper respiratory tract obstruction. Aspiration by way of tracheotomy is the most successful treatment of lower tract obstruction, providing postural drainage, bronchoscopic aspiration, and aspiration by other methods have not proved effective or feasible

An evaluation of the complications secondary to tracheotomy indicates that the procedure is not a hazardous one, but a relatively safe operation, offering many advantages to the patient.

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AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1 and continue for a period of ten months. Registrations must be completed before August 15. Detailed information and application forms may be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minnesota.

LOWER ESOPHAGUS PROBLEMS.*

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In considering lower esophagus problems this presentation is confined to a discussion of those symptoms caused by changes in the anatomy of the esophagus, diaphragm and cardia. Consideration of inflammatory lesions are limited to esophagitis seen in two cases, following left pneumonectomy and scleroderma of the lower esophagus, both suggesting a newer concept as to the etiology of esophagitis.

Bozer[1] in his article on "Endoscopic Problems" included an interesting excerpt from Thomas Willis'[2] book of 1679, describing the treatment for symptoms suggesting cardiospasm:

> "No less well, a very rare case of a certain man of Oxford shew an almost perpetual vomiting to be stirred up by the shutting up of the left orifice. A strong man, and otherwise healthful enough, laboring for a long time with often vomiting, he was wont very often, though not always, presently to cast up whatsover he had eaten. At length the distress having overcome all remedies, he was brought into that condition, that growing hungry he would eat until the esophagus was filled up to his throat, in the meantime nothing sliding down into the ventricle, he cast up raw (or crude) whatsoever he hath taken in: when no medication could help and he languished away for hunger, and every day was in danger of death. I prepared an instrument for him like a rod of whale bone with a little round button of sponge fixed to the top of it; the sick man having taken down meat and drink into his throat, presently putting down in the esophagus he did thrust down into the ventricle, its orifice being open, the food which otherwise would have come back again; and by this means he hath taken of his sustenance for 15 years and doth yet use the same machine and is yet alive and well,

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who would otherwise perish for the want of food. Without doubt in this case the mouth of the stomach always closed, either by a tumor or palsie, nothing could be admitted into the ventricle unless it were violently opened." This problem is still with us some 274 years later.

Since we are considering problems caused by changes in the structures around the hiatus for the esophagus in the diaphragm, a brief review of the anatomy is in order. From extensive origins about the thoracic cage muscular fibres converge to a common insertion to form the central tendon. The two principal openings through the diaphragm, in which we are interested, are the hiati of the esophagus and aorta. Both are behind the central tendon, the esophagus being surrounded by a sphincter-like arrangement of the crural fibers and the aorta just posterior to the middle arcuate ligament. The esophagus and aorta are incorporated by a figure "8" musculofibrous investment. There is no demonstrable muscular connection of the esophagus and diaphragm, these being fused by considerable amount of strong connective tissue. The sphincter cardia is inferior to the diaphragm and below the cardiac antrum. This anatomical factor will be dealt with later in showing the various epi-diaphragmatic intrusions of the sliding hiatal hernia, hiatal hernia, and parahiatal hernia.

The mechanics of respiration are important by reason of the variations of intra-thoracic pressure. During a respiratory cycle the maximum change in pressure is from — 70 to + 100 mm. of Mercury. At the time of a severe cough this pressure excursion is sudden and not over a normal four to five second cycle. The lung is invested with elastic fibers which deflate the lung to varying degrees, at the close of the positive pressure period. The positive pressure period is initiated by the inhibition of the diaphragm and intercostal muscles. Though the diaphragm is fixed to the vertebrae in the area of the central tendon during a coughing episode the stomach rises towards the esophageal hiatus with a water-hammer effect. This thrust on a weak or malformed hiatus is the cause of hiatal and parahiatal herniations.

Evans'[3] discussion on "Sliding Hiatal Hernia" has brought out some interesting anatomical findings of the lower esophagus, as seen on barium studies. Here we see a diagrammatic representation of the lower esophageal segment and esophageal hiatus. Dr. John A. Evans has kindly given me permission to use his illustration.

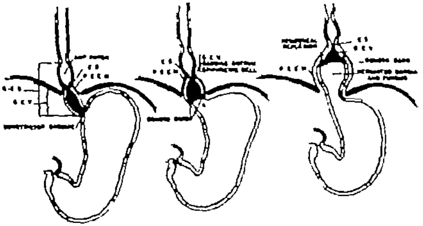

Fig 1.

G.E.V. Gastro-esophageal vestibulo-cardiac antrum
G.E.S. Gastro-esophageal segment of expulsion.
I.E.S. Inferior esophageal sphincter
P.E.E.M. Phreno-esophageal elastic membrane

1 Relationship of the normal hiatus. Black area is the gastro-esophageal vestibule

2 Relationship of the so-called hiatal insufficiency The blacked-in segment of the esophagus lying in and partially above the hiatus, represents the distended gastro-esophageal vestibule, cardiac antrum or so-called epiphrenic bell of Anders and Bahrmann It is this arrangement that is most difficult to interpret during Roentgen examination and must be differentiated from a small sliding hernia. During the roentgenoscopic phase of the examination this is frequently possible only by demonstrating the integrity or incompetence of the cardiac sphincter.

3 Diagram of sliding hiatal hernia in frontal plane showing the relation of the various structures.

From this excellent diagrammatic representation it is clear to see how any thinning or malformation of the esophagus hiatus and intra-thoracic and intra-abdominal pressures makes possible hiatal changes by the dilatating action of the gastro-esophageal vestibule. In similar manner a para-esophageal herniation may occur through and along the hiatal connective tissue of the diaphragm.

Esophagitis, or better reflux esophagitis, occurs only when the sphincter is insufficient, and free hydrochloric acid is present. This condition has been attributed to inflammation and vagal and phrenic interruptions. It is the purpose of this paper to present two different types of cases to suggest another or newer cause of reflux esophagitis.

This is the case of an adult with a left pneumonectomy for a benign adenoma of the left bronchus. Postoperative course was uneventful. This was done four and one-half years ago, and at the time of operation there was no interruption of the vagus or phrenic nerve. For the past two years the patient has had progressive episodes of heartburn and retro-sternal distress, which were relieved by medical treatment. Examination by X-ray shows this interesting picture of the esophagus, stretched, angulated and fixed to the latero-posterior parietal pleura by fibro-thorax. Esophagoscopy was not attempted. The lateral angulation and stretching has pulled the esophagus in such a way that the sphincter is no longer in a plane, but has incompetent abutting of the sphincter muscle; rather it is a lipping action, allowing some seepage of gastric contents.

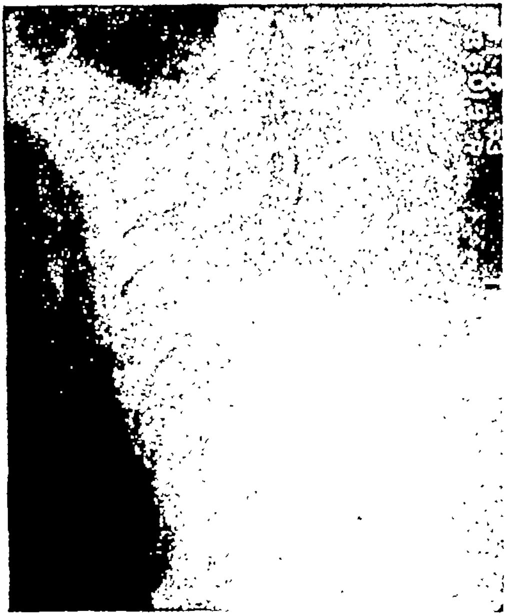

Fig. 2.
P. A. Film of Chest.

The second case is that of a 40-year-old white female with a four-year history of scleroderma of the esophagus, proven by biopsy. For the past eight months she has had regurgitation, burning sensation and difficulty in swallowing. The stomach had free hydrochloric acid. Fluoroscopy failed to reveal any peristalsis; there was fixation of the musculature and sufficiency of the sphincter.

Fig. 3

This picture shows the scleroderma taken four years ago, showing narrowing of the esophagus and dilatation of the proximal esophagus. A follow-up picture shows the recent presence of an ulcer caused by the esophagitis.

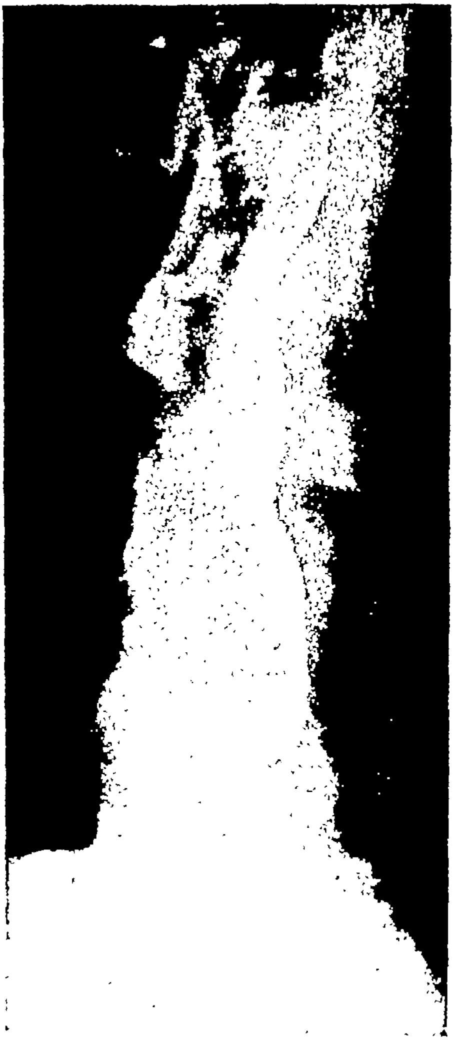

Fig. 4.

These two varied cases have developed esophagitis secondary to a lesion proximal to the lower esophagus, in each of which there was no interruption of the vagus or phrenic nerve. It is difficult to show the pathology causing esophagitis before the patient comes to us with symptoms referable to the condition. Reflux esophagitis occurs only when the sphincter is insufficient and free hydrocholic acid is in the stomach. In these cases we have seen, the sphincter was made insufficient by traction in the man who had a pneumonectomy, and in the woman who had muscular displacement from a sclerotic lesion causing sphincter insufficiency.

It is difficult to imagine that a generalized inflammation of the sphincter area would cause reflux esophagitis, since the edema would be equal throughout this area. It seems more logical that lesions such as a tumor mass, an eroding carcinoma, a granuloma, or fibrotic lesion would cause distortion of the sphincter plane, aside from proximal lesions.

Concering the problem of esophageal diverticula, especially the epiphrenic type, Putney, et al.,[4] have brought out several etiological suggestions. In considering the non-traction type, it is interesting to note the work done by Strauss and Lubitz,[5] showing the glandular structure of the stomach, cardia and esophagus. Unlike the stomach and cardia, the esophageal glands are racemose and are found beneath the muscularis mucosa. This could cause a weakness in the esophageal wall, and any increased intra-esophageal pressure over a period of time would cause a herniation of the wall and form a diverticula, or diverticulae. Dilatations of these glands are common, and at times reached cystic proportions.

SUMMARY.

Problems of the lower esophagus have been presented on the basis of anatomical changes of the esophagus, diaphragm and cardia. The problem of reflux esophagitis has been considered, and believed to be caused by alterations of the sphincter plane, by local and proximal lesions. It is suggested that diverticula of the esophagus is caused by the glands being under the muscularis mucosa and causing weakness of the wall.

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CLINICAL OBSERVATIONS ON END-ORGAN DEAFNESS.*

A Correlation with Cochlear Anatomy.

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The concept that all cases of nerve deafness must inevitably progress on a predestined course is no longer tenable. Recent observations by Hilger, Goltz[1] and Williams[2] suggest that there is hope of salvage in some instances, if cases are individualized and etiological aspects are considered on a physiological basis, before irreversible changes occur in the inner ear. Pathological changes in the cochlea, which result in hearing impairment, gradually become irreversible. Therapeutic failures in the past have resulted from attempting to reverse advanced cochlear pathology, and relying solely upon the use of drugs for which the pharmacological effect was at best somewhat uncertain, as far as the ear was concerned.

Exact knowledge concerning the histopathology of early endorgan deafness is very limited, because the organ of Corti undergoes lytic changes soon after death; furthermore, current histologic technique alters the cochlear structures—in particular, the normal contact of the tectorial membrane with the hair cells and their supporting structures. Recent studies by Hilding[4] have confirmed the opinion expressed by Shambaugh, Sr.[3] in 1907, that the tectorial membrane in the living state is attached to the surface of the organ of Corti. The possible clinical significance of this relationship has been discussed by Hilding[5] and will be further considered here.

In this discussion some cases will be cited; first, to illustrate significant improvement in auditory threshold, believed to have resulted from therapeutic measures. Second, some

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possible correlations between these clinical observations and known anatomical facts, together with theoretical considerations will be considered.

Bone conduction threshold and tuning fork tests were done on all cases, and established a diagnosis of nerve deafness in each. Only air conduction thresholds are shown in order to simplify the charts. The Eustachian tubes were inflated and found to be patent in all cases. These particular cases were selected because they represented a cross-section of the clinical problems encountered in managing the phase of otology under discussion.

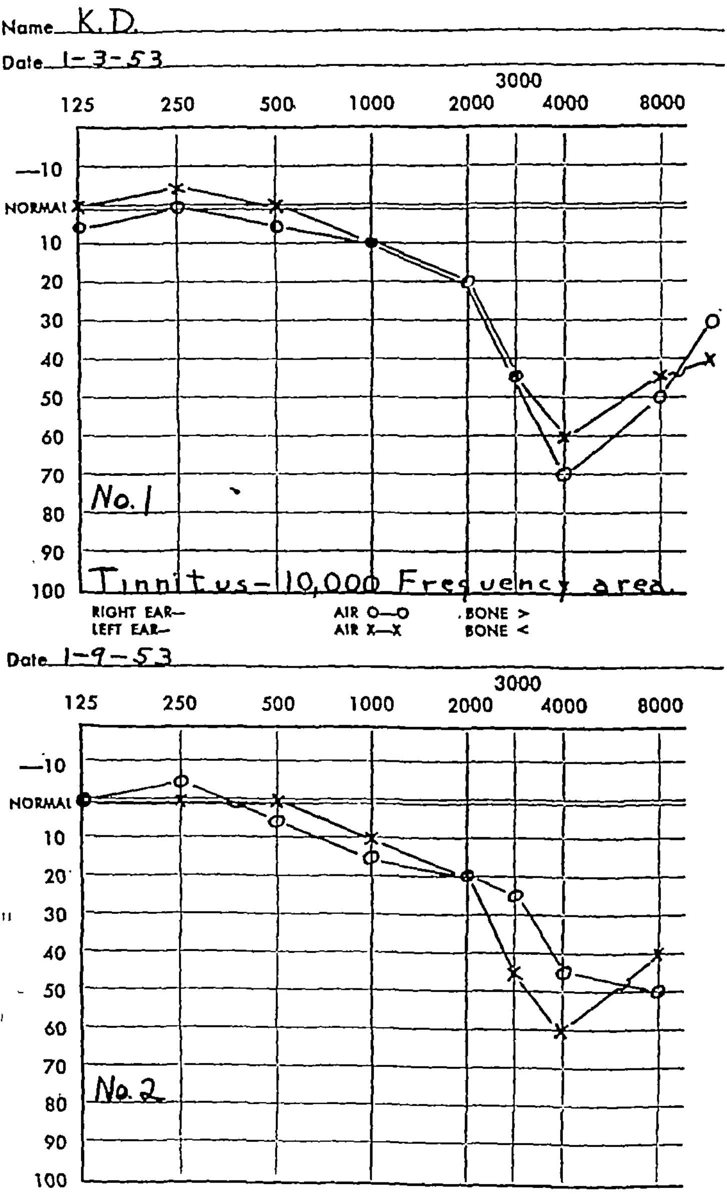
Case 1. K. D., Age 20. Tinnitus was first noted four months ago following a cold. It was more noticeable in the right ear. Indefinite vertigo had been present for six weeks. A feeling of pressure in the ears and an increase in tinnitus preceded the episodes of vertigo The radio sounded hollow and out of tune and had an echo-like quality. There were numerous gastrointestinal and chest symptoms which an internist concluded were due to autonomic instability. Patient smoked two to three packages of cigarets daily. There was no history of acoustic trauma. Dissatisfaction with the school he was attending and indecision as to a life vocation seemed to be the immediate basis of his psychosomatic disturbance.

Examination revealed normal tympanic membranes. Vestibular response to cold caloric stimulation was normal in both ears. He was given banthine and roniacol and advised to quit smoking. The tinnitus and vertigo gradually diminished and there was some subjective clearing of his hearing. It was believed that his inner ear upset was the result of his psychosomatic disturbance and excessive smoking. It will be noted in audiogram No. 2, Fig. 1, that there has been partial reversal of the audiometric notch in the right ear, but the left ear lesion had apparently progressed beyond the stage of reversibility.

Case 2. M. J., Age 27. A slight hearing impairment in the right ear had been present for two years following a suppurative otitis media. The patient was a surgical nurse, and in recent months there had been a lack of clearness in the left ear which caused some difficulty in understanding the mumbled words of surgeons. She smoked one package of cigarets daily. There was no tinnitus.

Examination revealed a large central perforation of the right tympanic membrane. The left tympanic membrane was normal. The audiometric curve reveals a notch at 2000, and a high tone loss in the left ear (see No. 1, Fig. 2). She was advised to quit smoking and was given roniacol by mouth. Three weeks later there was both subjective and audiometric improvement of hearing in the left ear (see Audiogram No. 2, Fig. 2).

Case 3. W. S. B., Age 39. Some hearing loss for high-pitched voices had been noted recently, especially in the left ear. There was some high pitched tinnitus, more noticeable in the left ear. A chronic postnasal discharge of variable degree had been present for several years. Five years previously the patient had had a temporary hearing loss following an upper respiratory infection. There was no history of acoustic trauma. He smoked about one package of cigarets daily.


Name K.D.
Date 1-3-53
125
250
500
1000
2000
3000
4000
8000
—10
NORMAL
10
20
30
40
50
60
70
80
90
100
No. 1
Tinnitus - 10,000 Frequency area.
RIGHT EAR—
LEFT EAR—
AIR O—O
AIR X—X
BONE >
BONE <
Date 1-9-53
125
250
500
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—10
NORMAL
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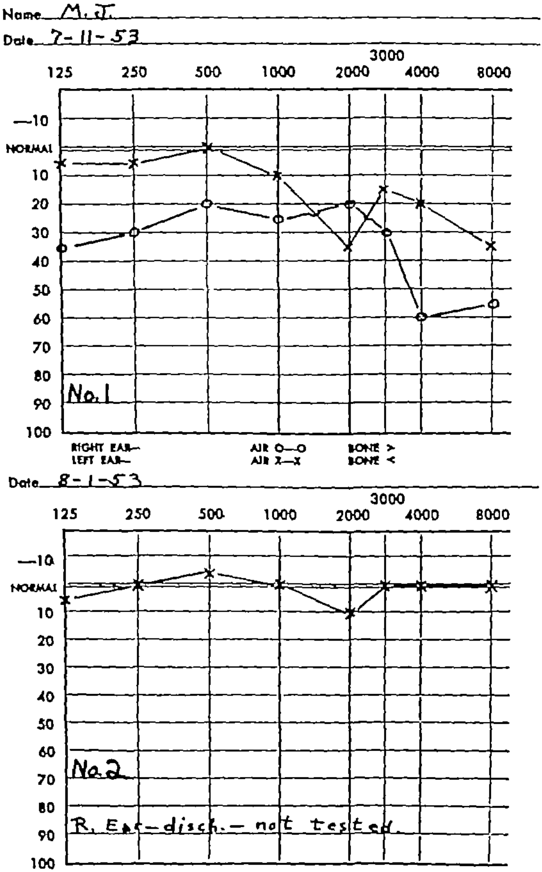
100
No. 2

Fig. 1.


Name M. J.
Date 7-11-53
125
250
500
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2000
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4000
8000
—10
NORMAL
10
20
30
40
50
60
70
80
90
100
No. 1
RIGHT EAR—
LEFT EAR—
AIR O—O
AIR X—X
BONE >
BONE <
Date 8-1-53
125
250
500
1000
2000
3000
4000
8000
—10
NORMAL
10
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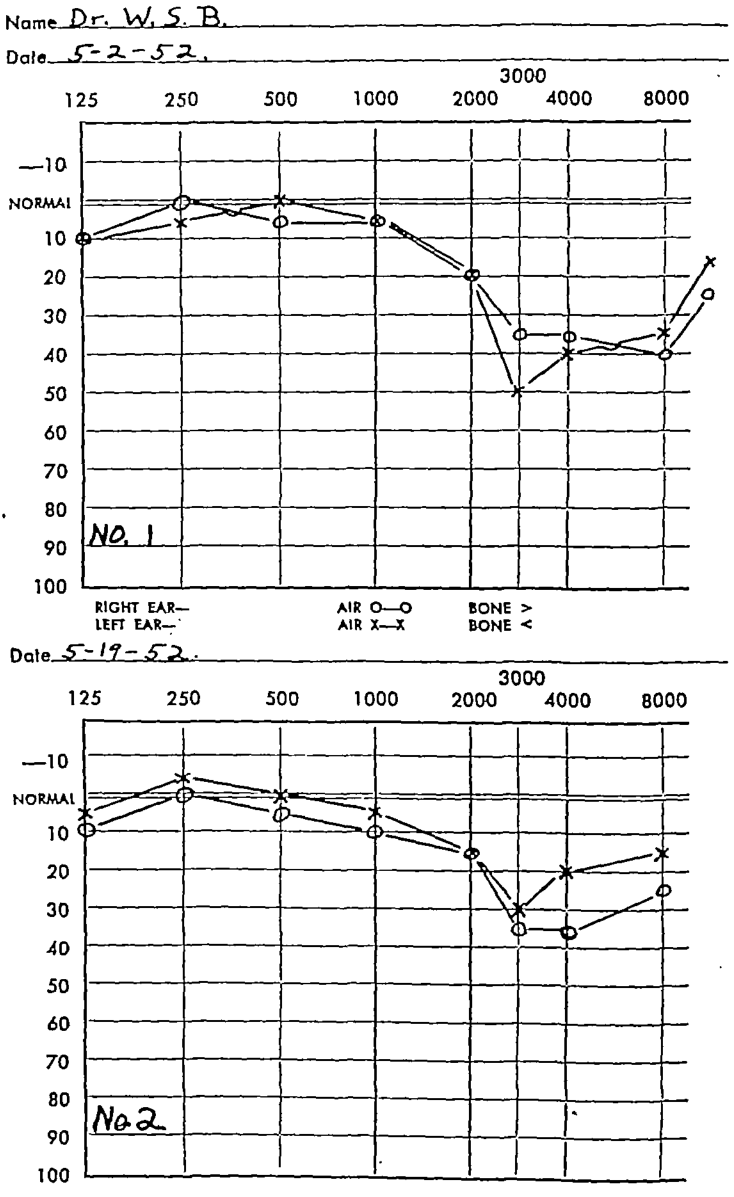
40
50
60
70
80
90
100
No. 2
R. Ear—disch.—not tested.

Fig. 2.



Name Dr. W. S. B.

Date 5-2-52.

Date 5-19-52.

Fig. 3.

On examination both tympanic membranes were normal. There was a moderate amount of mucoid secretion in the nasopharynx, and some scattered follicles of lymphoid tissue were present. The sinuses were clear on transillumination. The initial audiogram shows a moderate high tone loss (see No 1, Fig 3). He was advised to quit smoking and was given roniacol There was gradual subjective and objective improvement in hearing A marked decrease in postnasal drainage was noted. The hearing improvement occurred chiefly for the high tones in the left ear (see No 2, Fig. 3).

Case 4. F. G., Age 30. This patient was concerned chiefly with tinnitus following recent exposure to gunfire He had noted tinnitus previously after hunting, but it had always ceased after a few days He was not aware of any hearing impairment There was a history of fall hay fever and perennial vasomotor rhinitis of moderate severity

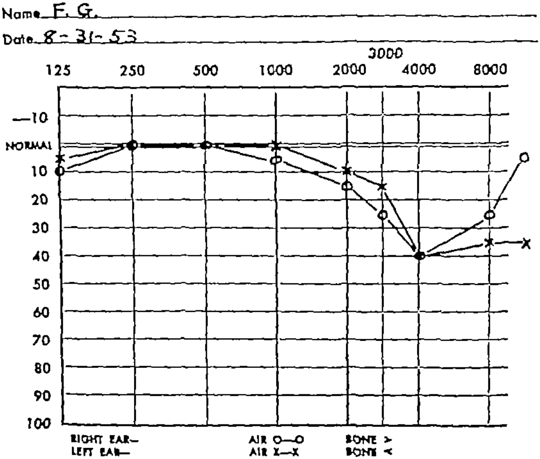


Fig 4.

Both tympanic membranes were normal Audiometric examination revealed a typical acoustic trauma notch at 4000 (see Fig 4). He was advised to discontinue smoking and was given nitocinic acid by mouth. Ear plugs were provided for protection against future acoustic trauma. He did not return for follow-up study

Case 5 T J. N., Age 36 Attacks of vertigo, together with hearing impairment in the right ear, had been present for six weeks. These symptoms initially followed a rhinitis. Music seemed distorted Intermittent tinnitus in the right ear was present

On examination, the tympanic membranes were normal and the Eustachian tubes inflated readily. Audiometric examination revealed all of

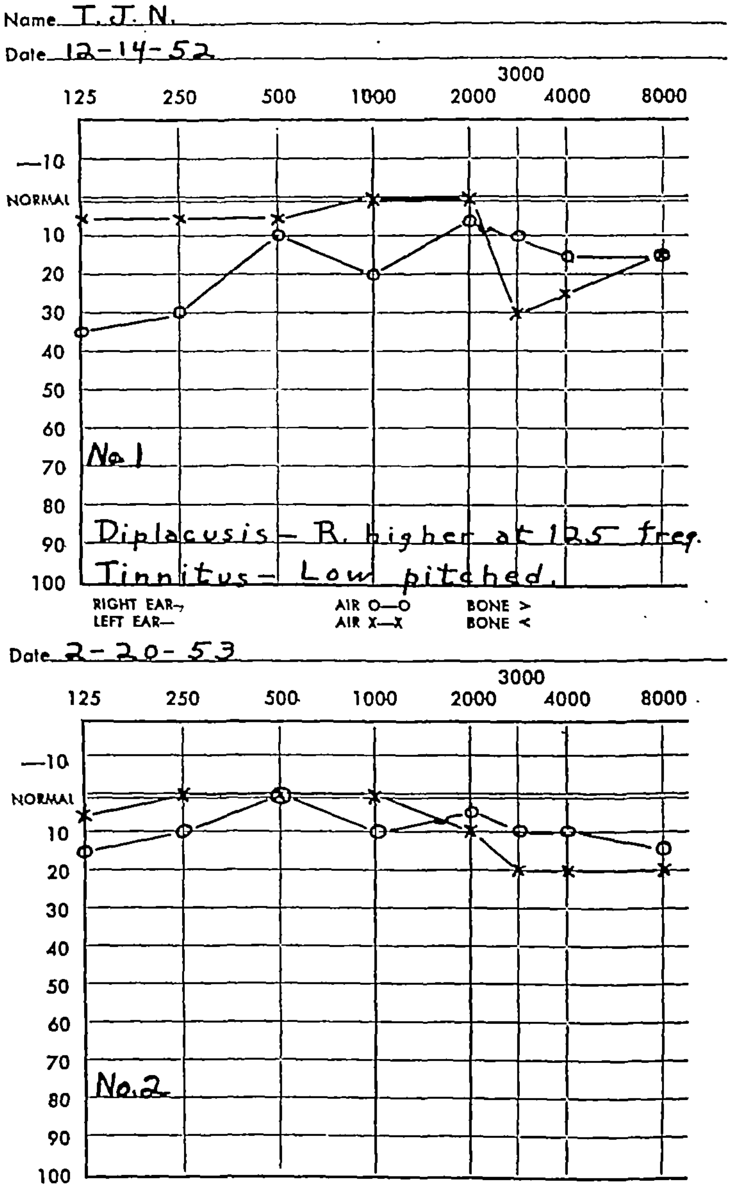

Name T. J. N.
Date 12-14-52
125
250
500
1000
2000
3000
4000
8000
—10
NORMAL
10
20
30
40
50
60
70
80
90
100
No 1
Diplacusis – R. higher at 125 freq.
Tinnitus – Low pitched.
RIGHT EAR
LEFT EAR
AIR O—O
AIR X—X
BONE >
BONE <
Date 2-20-53
No. 2

Fig. 5.

the characteristic findings of an early endolymphatic hydrops (see No. 1, Fig. 5). The patient was placed on a low salt diet and given nicotinic acid and potassium chloride by mouth. There was a gradual decrease in tinnitus and vertigo, and the hearing gradually returned to near normal (see No. 2, Fig. 5). The diplacusis disappeared as the hearing improved.

Case 6. N. C., Age 27. Tinnitus and deafness were first noted in the left ear four years ago. More recently there had been deafness in the right ear. There was some fluctuation of hearing at the onset, but none in recent months. Considerable mucoid postnasal discharge occurred in the morning. An episode of sneezing was experienced once or twice a week. Radium therapy to the nasopharynx had been applied with no benefit. The patient had been advised to obtain a hearing aid, but had not done so. Numbness of the fingers was frequently noted when doing such work as knitting.

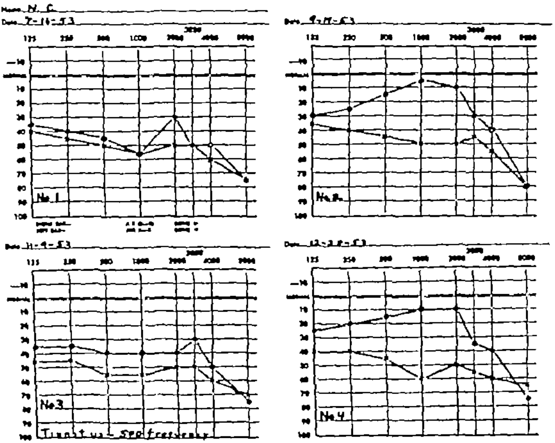


Fig. 6.

On examination, both tympanic membranes were normal and the Eustachian tubes inflated without difficulty. There was an excess of mucoid secretion in the nasopharynx. A smear of this secretion contained many polymorphonuclear cells and bacteria but no eosinophiles. The sinuses transilluminated clearly. The initial audiogram revealed a moderate degree of loss (see No. 1, Fig. 6). Speech tests revealed poor discrimination, especially in the left ear, and the narrowed dynamic range characteristic of recruitment.

The patient was advised to quit smoking and was given nicotinic acid by mouth. On this treatment there was a gradual improvement in hearing in the right ear and a noticeable decrease in postnasal drainage during the warm dry summer months (see No. 2, Fig. 6). Smoking was not entirely stopped but was decreased by about half. In the fall there was again a decrease in hearing (see No. 3, Fig. 6). The increased stress of weather variations and attending night school, in addition to her daily work as a medical technician perhaps contributed to this hearing decrease. Allergic studies cast some suspicion on milk. It will be noted that there was no improvement in hearing in the left ear, which was initially involved before the right. Evidently the changes in the left ear had progressed to a state of irreversibility. The last audiometric test is shown in No. 4, Fig. 6. The improvement in the right ear followed a more rigid avoidance of milk.

COMMENT.

The question might be raised as to whether the tonal dips and audiometric configurations in Cases 1, 2 and 3 represent pathology in the end-organ or in the spiral ganglion. It is generally agreed that the pathology of acoustic trauma is in the end-organ. Case 4, from a clinical standpoint, would be considered as one of acoustic trauma. There is no history of acoustic trauma in Cases 1, 2 and 3; however, the similarity of the audiometric curve of these three cases to Case 4 would certainly justify the assumption that the pathology in all three might be in the end-organ.

A diagnosis of endolymphatic hydrops (Meniere's syndrome) was made in Case 5. It was included in this study in order to illustrate what is believed to be a different type of the end-organ deafness. Case 6, from a clinical standpoint would be a case of cochlear hydrops, in view of the presence of considerable fluctuation of threshold and low-pitched tinnitus.

In most of the cases cited there was either a history of the ear symptoms starting after some type of rhinitis, or of excessive postnasal discharge at the time of examination. This relationship gave rise to the older concept of catarrhal deafness; however, rather than representing a cause-effect relationship between the nasal symptoms and ear disturbance, the nasal and ear symptoms may represent a simultaneous clinical expression of a basic neurovascular and neurosecretory disturbance in two areas of the autonomic and vascular distribution.

For this reason, it is necessary to consider the therapeutic approach beyond the confines of the nose and nasopharynx, and analyze each case individually, as to the many environmental and intrinsic factors which contribute toward the basic physiological disturbance.

An accurate evaluation of therapy in hearing impairment, believed to be due to end-organ changes, is extremely difficult because in some instances there is early reversibility even though no therapy has been applied. This is readily understood when we consider the diversity of etiological factors which may vary from emotional and psychic stress to definite infection in the upper respiratory passage. These etiological factors may singly or in combination express themselves in a final common pathway of arteriolar-capillary-venular dysfunction with consequent local electrolyte and nutritional disturbances involving the structures concerned in inner ear physiology. The physiological and hypothetical aspects of this problem have recently been comprehensively and aptly covered by Hilger[1] and Williams[2] and need not be considered in detail here.

Loch[6] studied tonal dips in 1365 school children, and found dips in 15 per cent of the boys and 5 per cent of the girls. He classified the dips into persistent, temporary and recurrent. Persistent dips were noted most frequently at the 4096 frequency and temporary ones were more often observed in the 10,321 area. About two-fifths of all dips were permanent. We can assume that the temporary dips disappeared because the etiological factor was corrected, either spontaneously or as a result of definite therapy. The permanent dip must be accepted as an irreversible lesion, or as an instance in which appropriate therapy was not applied. This study would certainly suggest that a tonal dip must not always be accepted as being inevitably permanent until a reasonable attempt has been made to uncover possible etiological factors and until indicated therapy has been applied; however, this must not be construed as an invitation toward unreasonably long and expensive treatment. Destructive surgical attack on questionable foci of infection is certainly to be condemned.

Wever[7] studied the Hopkins temporal bone sections concerning the problem of tonal dips and found no consistent pathological changes; however, he stated that "failure to find a lesion in a particular region does not signify that the tissues there were operating in a normal manner. There is no certainty that functional impairments are revealed by the histological technics used."

He observed that deafness in general, especially abrupt high tone loss, was noted more often in men. In speculating as to the basis of occurrence of tonal dips more frequently in the 4096 area, he considered two possibilities; first was the fact that the 4096 frequency lies in the region of resonance of the ear; hence this area is probably subjected to more mechanical disturbance. Second, he believes this area to be subject to more autolysis postmortem, possibly as a result of more susceptibility to physical breakdown in life.

An interesting feature of the anatomy of the cochlea is the constriction of its bony wall at the approximate region of perception for the 4096 tone (see Fig. 7). Larson[8] has listed this as one of the possible reasons why the initial loss secondary to acoustic trauma in general is greatest for the 4096 frequency. It is obvious that the sound pressure incidental to each cycle with a frequency lower than 4096 must pass through this "bottleneck." This fact, together with Wever's statement that the region of resonance of the ear is at the 4096 frequency, would mean that the cochlear structures in this area must be subjected to considerably more mechanical stress than in any other area.

There has been a marked renewal of interest in the tectorial membrane since the recent studies made by Hilding.[4] The usual textbook picture showing the membrane separated from the hair cells and their supporting structures is an artefact of tissue fixation and dehydration. In fresh specimens the tectorial membrane is very intimately bound to the organ of Corti. Hilding has incorporated this relationship into a theory of sound perception. Borghesan[9] has furnished further histological evidence which substantiates Hilding's findings and goes on further to state that he believes that the hairs from the hair cells are continuous with and, therefore, constitute the fibrils of the tectorial membrane. The fibrils are imbedded in an amorphous mass of gelatinous substance, which in the living, is almost fluid because of its high water

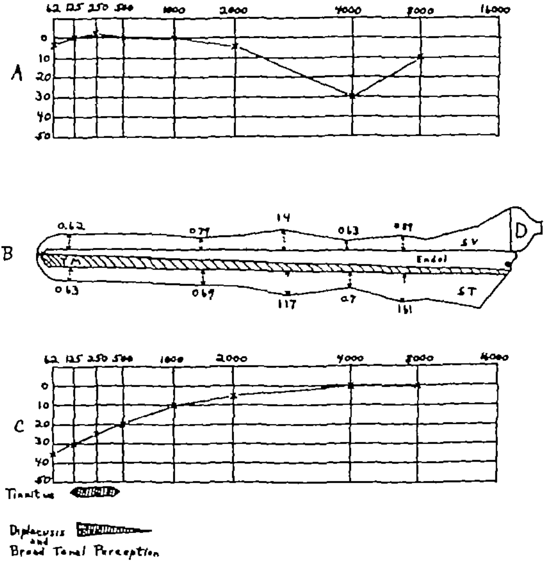


Fig. 7

A. An audiometric chart modified so that the position of each frequency on the chart corresponds to its approximate area of tonal perception in the cochlear sketch below in B. The audiometric curve illustrates a typical 4000 notch.

B. This is a sketch of cochlear dimensions taken from Fowler's loose-leaf "Medicine of the Ear. I have modified the sketch by inserting the endolymphatic duct and the tectorial membrane. The relative size of the tectorial membrane and the endolymphatic duct are schematic from descriptions in the literature and must not be accepted as representing exact relationships. Points to be noted are the constriction of the cochlea at the 4000 frequency area and the gradual increase in size of the tectorial membrane from the base to the apex.

S. V—Scala vestibuli
Endol—Endolymph
H—Helicotremma.
S. T—Scala tympani
T M—Tectorial membrane.

The numbers on the outside indicate the cross section area of the adjacent scala vestibuli and scala tympani in MM^2

C. An audiometric chart arranged as in A with a curve characteristic of cochlear hydrops. The tinnitus diplacusis and broad tonal perception are indicated as occurring in the low frequency area.

content. He believes that the shrinking of the tectorial membrane incidental to the usual histological preparation tears the hairs just above the cell itself.

The interposition of the tectorial membrane between the main body of the endolymph and the organ of Corti would necessitate diffusion of endolymph through the membrane in order to reach the hair cells and their supporting structures. Such a function was ascribed to the tectorial membrane by Richenbacher[10] in 1901.

Embryologically the tectorial membrane arises from the same epithelial ridge that forms the organ of Corti (Prentiss[11]). This might be considered as lending further credence to Borghesan's concept that the fibrils of the membrane are continuous with and actually a part of the hair cells.

What, then, is the significance of this possible additional aspect of tectorial membrane physiology to the question of tonal dips? Since it seems likely that the 4096 area is subjected to more mechanical stress than other areas, the fibrils at the point of junction of the main tectorial mass and the hair cells will be traumatized and possibly torn more readily in this area than in the rest of the cochlear duct. A mild degree of fibrillar damage could be reversible but perhaps a more severe degree would not be. The effect of such damage would be twofold: first, there would be interference with the mechanics of sound perception in this area; second, the nutrition of the hair cells would be impaired.

If this point of constriction is significant, then variations in the degree of constriction could account in part for variations in susceptibility to acoustic trauma and non-traumatic early physical deterioration in this area, with a subsequent tonal dip. Any minor disturbance in nutrition of the organ of Corti in general, which otherwise might not lead to impairment of cochlear perception might, in the presence of a greater degree of cochlear constriction in this area, result in definite localized diminution in hair cell perception.

THERAPY.

The rationale of therapy applied to the cases cited earlier is based largely on the concepts expressed by Hilger and

Williams. A small but definite percentage of individuals has an unquestioned harmful physiological response to the effects of smoking. It has been routine to advise patients to discontinue smoking during the immediate postoperative period after fenestration. On resuming smoking many of the patients note a definite increase in vertigo during the smoking of each cigaret. The fact that not all patients experience this reaction substantiates the idea that there is a wide individual variation in tolerance to nicotine. I believe that in most of the cases cited previously, smoking was a significant factor in the etiology of disturbed inner ear physiology.

Vasodilating therapy is given on the presumed presence of vasospasm of some portion of the inner ear vascular tree. Psychological stress in the form of interpersonal emotional problems, business or other responsibilities exceeding the individual's normal capacity, frustrations, etc., must be taken into consideration in proper perspective in the overall management of these cases.

The excessive use of caffeine in any form may be a contributing factor in etiology. The individual who fortifies himself with a stimulant several times a day whenever his energy lags may be "whipping a tired horse" and inviting eventual imbalance of the intricate and sensitive autonomic system.

The role of undue physiologic fatigue in initiating disease in general is too well-known to require further comment; however, the importance of fatigue to the point of exceeding one's physiological limits is too often neglected in counseling our patients in this age of stimulants and sedatives, histamine and antihistamines, vasoconstrictors and vasodilators, etc. Unfortunately, too many patients insist on pursuing ways and habits of living which for some can eventually lead only to a state of hopeless autonomic confusion. They seek to obtain refuge from their ultimate fate of physiological disorganization by consuming multiple boxes of pills rather than by reestablishing a more hygienic mode of life.

Allergy is another aspect of etiology which must be given proper consideration. Jordan[12] has reported a few instances of tinnitus due to proven food allergy. Tinnitus is frequently the earliest symptom of impending cochlear deterioration. The allergic problem is definitely interwoven with that of autonomic dysfunction, as is illustrated by Case 6.

The relationship of acetylcholine release or formation incidental to trauma in a closed space is of theoretical interest. Increased free acetylcholine has been found in the spinal fluid of animals subjected to experimental cerebral trauma.[13] On the basis of this study, large doses of atropine have been used with some benefit in treating selected cases of cerebral trauma in humans.[14]

If the concept of mechanical stress incidental to the 4096 cochlear constriction is valid, it is conceivable that repeated excessive local liberation of acetylcholine could increase capillary permeability sufficiently to initiate a cycle of arteriolar-capillary-venular dysfunction. This would eventually predispose the area to autonomic imbalance and its consequences, whenever any one or a combination of several of the previously discussed etiological factors became operative.

In other words, a small segment of the vascular bed, which has previously become conditioned to respond in a stereotyped manner to local trauma, under conditions of general stress may react to a degree that proves detrimental to the local structures. The individual capacity to withstand stress varies greatly, and may account for the fact that the same etiological factors which will produce inner ear disturbances in one individual are well tolerated by others.

Endocrine balance is intimately related to autonomic function and must be given proper consideration, as indicated by the history and clinical picture.

ANATOMICAL CONSIDERATIONS.

The possible significance of the constriction of the bony wall of the cochlea in the region of perception for the 4096 frequency has already been discussed. The degree of variability of this constriction needs to be studied further. Minor disturbances in the integrity of the tectorial membrane may prove to be important in explaining the cause of perceptive lesions for which to date there has been no visible anatomical basis on microscopic study. Once the physical continuity of the tectorial membrane, with its attachments, has been disrupted in a small area, continued mechanical stress will undoubtedly spread this disruption in much the same manner as a strong wind would extend a small tear in a sail.

The inner ear disturbance in Case 5 is due to endolymphatic hydrops. The cochlea of the involved ear (right) presents all the early findings characteristic of this condition, *i.e.*, greater loss for the lower frequencies and a coarse broad character of tonal perception for the lower frequencies. It will be noted that the early symptoms all occur in the apical area of the cochlea (see Fig. 7-C). If the deafness continues to progress, the loss eventually involves the entire audiometric scale.

Examination of the relative dimensions of cochlear structures reveals that the tectorial membrane is much larger in the apex than in the base. The scala media, on the other hand, is slightly smaller in the apex than in the base. Unfortunately, current histological techniques do not permit any conclusion as to the state of the tectorial membrane in endolymphatic hydrops before the artefact of fixation and dehydration is produced.

The fibrils of the tectorial membrane are imbedded in an amorphous mass of protein-like substance, which is no doubt altered in physical characteristics, and possibly size, as a result of the altered character of the endolymph in an ear with hydrops. In all probability, any appreciable edema of the membrane would change its physiological response to sound more in the apex than in the base because of the greater size of the membrane in the apex. Shambaugh, Sr.,[3] first suggested that the tectorial membrane changes mights account for such cochlear symptoms as diplacusis and some instances of tinnitus. Mygind[14] has more recently mentioned tectorial membrane changes in labyrinthosis.

Cook[14] has noted that patients who clinically have cochlear hydrops are unusually susceptible to acoustic trauma. Relief of the hydrops by medical measures enabled a number of such individuals to return to working in a noisy environment. We might speculate that the edema of the tectorial membrane created an unusual tension on the hair cells insertion into the membrane. As the edema subsided this abnormal relationship was corrected, and as a result greater tolerance to intense sound returned. Cook's observations might suggest some relationship between recruitment and changes in the tectorial membrane.

The question as to what happens in the fluid-containing spaces within the organ of Corti in cochlear hydrops still remains to be answered. Another question posed is why, in the presence of seemingly similar etiological factors, does the cochlea in one individual respond with a clinical entity interpreted as cochlear hydrops, and another with a tonal dip? The answer may very well lie in variations in anatomy such as areas of cochlear constriction and tectorial membrane size. General individual physiological deviations involving capillary permeability and electrolyte balance may predispose some ears to an edema-type of response in the presence of autonomic dysfunction. Pearlman, et al.[17] have recently pointed out the limitations of our present knowledge concerning the relationship of electrolyte balance and Meniere's disease.

SUMMARY.

1. Several cases are cited in which reversal of an early end-organ deafness was observed. The therapeutic approach applied to these cases was relatively simple.

2. Failure of both ears to improve indicated that the lesion had progressed to a stage of irreversibility in one ear.

3. Enthusiasm for the efficacy of a given therapeutic measure must always be tempered with the possibility that there is in some instances a spontaneous reversal of the lesion.

4. A broad physiological approach to a wide range of etiological factors is perhaps the most important aspect of the general problem of early nerve deafness.

5. Theoretical correlations have been made concerning the possible relationship of some features of cochlear anatomy and clinical observations on nerve deafness.

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THE ANTIBIOTICS AS AN ADJUNCT IN THE TREATMENT OF OTOLARYNGOLOGIC DISEASES.*

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In recent years the antibiotics have been introduced into our armamentarium to combat infection as a *deus ex machina,* so sensational has been their effectiveness. It is a precarious venture to enter into a primary dissertation on the antibiotics before an audience of this caliber. I must, therefore, limit my comments to a few remarks in order to refresh our memories and to bring us onto a common ground for purposes of discussion. The picture changes so rapidly that categorical statements are futile, since a certain amount of confusion and lack of sequence is inevitable.

From the very comprehensive treatise on *Antibiotic Therapy*[1] it may be of historical interest to quote the following paragraph:—

"The term 'antibiosis' probably was used first by Vuillemin[2-1] in 1889 to describe what we consider to be 'the survival of the fittest', whereby a creature destroys the life of another to preserve its own. The use of the word 'antibiosis' to describe microbial antagonism was adopted by Marshall Ward ten years later.[3-1] It was not, however, until 1942 that Wakeman[4-1] proposed the use of the term 'antibiotics' to define those chemical substances of microbial origin which possess anti-microbial activity. With the increasing search for new antibiotics, this term has been enlarged to include not only chemical substances of microbial origin, but also those of plant and even animal tissues. In any case, the word antibiotic has now become firmly established, not only in the

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minds of scientists throughout the world, but in the lay mind as well, through the tremendous amount of publicity these drugs have received during the past ten years. Notwithstanding the fact that the term antibiotic is misused in some instances, and that it may not be the best word to define this important group of substances, it is unlikely that it can be displaced by another of perhaps greater merit, such as 'antimicrobial,' 'antagonist' or 'bacteriostat,' because it has become so firmly fixed in both the scientific and lay literature."

Under the pressure of our daily routine we may be prone to accept the antibiotic as an omnipresent umbrella to tide us over the worst cloudbursts of our clinical storms, with the hope that we may avoid the devastating inundations of the more serious and intractable infections that bring havoc to the suffering patient. It may be, that at times, we do not give sufficient thought to the serious reactions of an antibiotic in our efforts to accomplish the primary salutary effects.

It is well to jog our memories to recall the basic principles of resistance to infections as predicated by the antibody reaction of the individual. The mechanism underlying recovery in all infections is essentially anti-bacterial in character, brought about by the development and the actions of specific anti-bacterial antibodies, together with the activity of the phagocytic cells. This bio-chemical response to infection is still recognized as the all-important factor in preventing and limiting bacterial invasion.

It is not amiss to state that only too frequently this balance is disturbed by the premature administration of antibiotics, thus depriving the body of an adequate stimulus for the formation of antibodies and resulting in a pseudo-control of infections, with subsequent exacerbations because of a temporary and inadequate bacteriostasis of pathogenic microorganisms. The cardinal principle that resistance to infection is natural, or/and acquired, and that acquired resistance is the result of minimal or manifest infections, is an accepted theorem.

In otolaryngology, the antibiotics in common usage have been Penicillin, Aureomycin, Terramycin, Chloromycetin, Streptomycin, Dihydrostreptomycin, and recently Ilotycin.

Penicillin is primarily active against the gram-positive group of organisms, either as a bacteriostatic or a bactericidal agent. It is also active, but less so, against meningococci and gonococci in the gram negative group, while hemophilus influenzae is resistant to it.

While it was originally stated[1] that only certain strains of staphylococci became resistant to penicillin, Romansky[5] has brought out the fact that in the past few years investigations have shown that there is also occurring an increasing resistance of hemolytic staphylococcus aureus, hemolytic staphylococcus albus, non-hemolytic and alpha-hemolytic streptococcus (viridans).

Streptomycin and Dihydrostreptomycin. The chief concern of otolaryngologists in these antibiotics lies in their toxic reactions upon the internal ear, on the cochlear as well as the vestibular divisions of the VIIIth nerve. Streptomycin and dihydrostreptomycin affect both gram-positive and gram-negative organisms as bacteriostatic as well as bactericidal agents; however, the mycobacteria is the most important group of organisms acted upon by these drugs as bacteriostatic agents. They are ineffective against fungi, viruses and rickettsia. Hemophilus influenzae and pertussis are sensitive to their action. While these are drugs of supposedly low toxicity, para-aminosalicylic acid is being successfully used in combination to delay the growth of resistant strains of the tubercle bacillus and enhance the action of streptomycin.

Aureomycin, primarily bacteriostatic, is bactericidal in high concentrations. It affects both gram-negative and gram-positive organisms, also certain large viruses and rickettsia. These organisms do not easily develop resistance to the drug.

Terramycin. This drug has practically all of the characteristics of aureomycin, with the advantage of being less toxic and causing fewer and less severe side reactions.

Chloromycetin until recently was considered to be one of the most effective and least toxic of the broad-spectrum antibiotics at our disposal; however, in 1950 and subsequently, reports have appeared in the literature of blood dyscrasias, specifically aplastic anemias, which occurred following the administration of chloromycetin over varying lengths of time. Among these, several fatal cases were reported, one within two days of the first administration of the drug.[6] These dire results might have been anticipated, since Smadel in 1949[7] had sounded a warning that the presence of the nitrobenzene radical in chloromycetin constituted a "potential danger as a hematopoietic toxin." As a consequence, the use of this drug has now been practically discontinued.

Despite the unquestionable effectiveness of the antibiotics, their uses have been curtailed by the many toxic reactions that have ensued. Such reactions we have all seen. We know that penicillin may cause a black tongue, a glossitis or a stomatitis, various degrees of urticaria; also more serious anaphylactic and allergic reactions, or even a true Herxheimer reaction. These distressing and alarming sequelae may develop after one or many administrations, occur immediately, or two or three weeks after its use is discontinued. Severe reactions may be averted in patients who have been given penicillin previously, by prescribing it orally; this avoids the immediate "build up" that may follow intra-muscular injections, especially if coupled with antihistaminic therapy.

The toxicity of the broad-spectrum antibiotics is usually manifested by gastric symptoms, nausea, at times vomiting, and diarrhea. The diarrhea and anal or vulvar pruritis are the more frequent and the more severe results of sensitivity. The diarrhea is said to be caused by the bactericidal effect of the antibiotics on the normal flora of the intestinal tract and their replacement by the acid producing yeasts or fungi of the monilial family especially. Despite the frequently published reports that the incidence of this penomenon is an occasional complication, our experience has been to the contrary. In some instances it has been so serious that it has established a colitis and a pruritis ani that has persisted or recurred for two or three years afterward.

Recently it has been determined that some of the untoward side reactions may be avoided and greater effectiveness achieved by the synergistic action of one or more of the antibiotics, combined with each other, or with chemotherapeutic agents. This is notably true with penicillin and streptomycin, dihydrostreptomycin, or with the triple sulfonamides, and the use of the broad-spectrum antibiotics with the triple sulfonamides. We are all familiar with such combinations which have been made available by many of the manufacturers of these drugs. In some instances these seem to exert a greater antimicrobial activity on the border-line strains of certain organisms, or on those which may have become resistant to one or other of the drugs given singly; however, if signs of sensitization are manifest one is at a loss to know which drug may be the offender, if not both drugs simultaneously.

The question of organisms developing resistance to the various antibiotics and chemotherapeutic drugs has become a realistic fact, as more and more of these drugs are being used, and more accurate *in-vitro* experiments reported.

Romansky[5] states that recently many more gram-positive and gram-negative organisms have shown an increased resistance to the broad-spectrum antibiotics, as well as to penicillin. This is especially true in regard to streptomycin, the use of which has not only activated many resistant strains of various organisms, but has caused an enhancement of growth of tubercle bacilli, according to Speedlove, et al.[8] This phase of antibiotic therapy must lead one to conjecture on the significance of the passage of certain pathogens from one individual to another. Such pathogens, carrying an increased virulence as the result of animal passage, and an acquired resistance to antibiotics because of previous exposure, necessarily or otherwise, may be instrumental in causing the intractable spread of certain infections in a community, even to epidemic proportions.

The intelligent use of antibiotics when carefully chosen and distinctly indicated, despite the known possibility of more or less serious side reactions, is a justifiable and calculated risk; however, the haphazard use of these drugs or their various modifications in solutions for intranasal medication, or for ear drops, or as lozenges or ointments, with the knowledge that sensitizations may be imminent, and with no assurance of benefit to the patient, is contra-indicated. Likewise, the routine administration of penicillin as a pre-operative adjunct when infection is not present, and as a protection against a presumptive infection with gram-positive organisms, thus unnecessarily exposing the patient to serious sensitization, is no more rational

It is a generally accepted fact that the administration of the antibiotics should be governed by the sensitivity of the infecting organisms to the drug We are all familiar with the procedure of obtaining a culture of the pathogenic flora and determining their sensitivity by special cultural methods The time lapse for such a report from the laboratory is usually four to five days, a loss of valuable time, which may occur in the most critical period of a patient's illness.

Realizing this deficiency we have been conducting experiments in the Department of Otolaryngology of St Louis University at the Firmin Desloge Hospital Clinic, to determine whether it is feasible and accurate to obtain this same information in a shorter length of time *

Our plan of procedure has been as follows

1 Tryptose-broth, whole-blood-agar plates used

2 Culture taken from patient with swab, which is twirled in 8 cc of tryptose broth

3 Blood agar plates (3) flooded with 8 cc. of inoculated broth, the excess being removed with sterile pipette

4 Surfaces of these plates were then allowed to become comparatively dry, and the discs containing the antibiotics were placed on the surfaces with sterile forceps The sensitivities were routinely run against the following antibiotics in the disc concentrations indicated

Aureomycin	60 mcg	30 mcg	10 mcg
Chloromycetin	60 mcg	30 mcg.	10 mcg.
Terramycin	60 mcg	30 mcg	10 mcg
Penicillin	10 μ.	1 μ.	0 5 μ.

5 The inoculated plates with the discs were incubated in an inverted position for 24 hours, and the zones of inhibition measured with a millimeter rule, the width of the zone indicating the effectiveness of the inhibiting property of the antibiotic against the entire flora cultured.

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* Funds for these experiments were obtained as a grant from the "Stuever Fund" of the Department of Otolaryngology of the St Louis University School of Medicine

6. The individual colonies were then picked and planted in 3 cc. tryptose broth, incubated for 24 hours, and replanted on blood-agar plates with the antibiotic discs in place as in the 24-hour plate technique. After another 24 hours of incubation the zones were noted, measured and compared with those found with the respective antibiotics on the 24-hour plates.

In this manner we were able to verify the accuracy of the rapid method for determination of the sensitivities of the organisms growing in symbiosis, compared to the slower method of the reactions of the organisms growing individually.

This survey extended over a period of 12 months. The cultures were taken from the throat, nose or ears of unselected patients in the Firmin Desloge Clinic. In a total of 250 patients: 70 per cent were throat cultures, most of which were taken as part of our routine procedure the day before a tonsillectomy; 17 per cent were from the ears, and 13 per cent from the nose.

The pathology present is not pertinent to this report, although the organisms found were non-pathogens, as well as pathogens, of nine different varieties.

In the 24-hour mixed cultures the zones of inhibition corresponded to those in the pure cultures in 237 instances, or about 95 per cent. The zones about the two weaker disc concentrations were considered more significant than those about the strongest concentrations. As a rule, the zones were about three-fourths as wide in the mixed cultures as in the pure cultures in similar antibiotics, and in the majority of the plates penicillin was the least effective inhibitor in the mixed cultures.

Of the mycins, chloromycetin usually created slightly wider zones than aureomycin and terramycin, which were about equal. In the later months of the survey ilotycin was used, though not recorded here, and it was found to inhibit less strongly than aureomycin and terramycin.

The types of organisms inhibited corresponded in general to the accepted criteria for the individual antibiotics.

From these findings we felt justified in deducing that *in-vitro* sensitivity tests of the anti-microbial properties of aureomycin, chloromycetin, terramycin, and penicillin, against 24-hour plated cultures of the entire bacterial flora of a focus, is at least 95 per cent as accurate as such tests against the pure cultures, which require 72 to 96 hours to interpret, using the disc technique on blood-agar plates as specified above.

The advantages of the 24-hour method are obvious in clinic practice, whereby the antibiotic thus indicated may be given almost immediately, and modified at the end of the 72-hour period if it is found advisable to do so.

In reviewing the many disadvantageous factors which have developed in connection with the administration of the antibiotics we are forced to admit that they are not the "panacea" which they originally gave promise of being. The unpleasant, and at times critical, reactions cannot be ignored, even though they may be ameliorated by the use of the antihistaminic drugs. Likewise the antibiotic resistant organisms, which nullify the anti-microbial properties of these drugs, must be respected.

We as physicians must not lapse into the unpardonable attitude of regarding the patient as a "test tube." The progress of infection in relation to the resultant pathological picture in the sinuses, the larynx and the ears must be carefully observed and therapeutic measures modified or discontinued as the indications arise. I view with great alarm and the utmost apprehension the prospect of irreversible hyperplastic otitis media and future conductive deafness in children who have been given the antibiotics to control otalgia or otitis media without keeping the condition of the membrana tympani under close observation, performing a paracentesis when indicated, and also testing the hearing, as apparent cure of the otitis media ensues.

We have seen many patients with unresolved maxillary sinusitis causing recurrences and constitutional signs of focal infection resulting from inadequate or no local treatment in addition to routine antibiotic therapy.

Flagrant instances are occurring daily of patients "passing on" their antibiotics to other members of their families and friends to cure infections which they consider similar to their own. For this reason a patient should be told which antibiotic he is being given, the symptoms of the possible reactions, and cautioned not to take the responsibility of administering the medication to anyone else. This advice should be prefaced by questioning the patient about previous exposure to antibiotics, and the possibilities of his own sensitivity to them should be carefully discussed.

It is, therefore, more imperative than ever that the otolaryngologist should closely follow such patients, since the classical text book pictures of the symptomatology and the pathological findings are so greatly modified that he alone is qualified to evaluate these changes and to act accordingly. The indications for surgical intervention in sinusitis, otitis media and mastoiditis should be recognized, as a recrudescence will undoubtedly come about in the wake of the inevitable limitations to the uses of the antibiotics in otolaryngologic conditions.

SUMMARY.

1. The antibiotics aureomycin, chloromycetin, terramycin and penicillin as adjuncts in the otolaryngologic treatment of diseases of the ear, nose and throat, are discussed in relation to their indications, contraindications and undesirable toxic side reactions.

2. A rapid cultural method is presented for the determination of the sensitivities of the entire flora of the ears, nose and throat for these antibiotics, as a guide to their earlier, more accurate administration.

3. The case is presented stressing the importance of closer observation by the otolaryngologist of patients receiving antibiotic therapy, in the treatment of pathological conditions in his field.

CONCLUSIONS.

1. The antibiotics cause certain undesirable side reactions in human beings: these may be definite contra-indications to their administrations.

2. Different varieties of bacteria are showing greater resistance to antibiotics, which render their administration futile in certain instances.

3. The 24 hours cultural sensitivity tests appear to be 95 per cent as accurate as the 72- to 96-hour methods, as indicators for the administration of the antibiotics.

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A CASE OF ATYPICAL OR QUESTIONABLE MASTOIDITIS IN A THREE-MONTH-OLD INFANT.*

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G. D., a three-month-old infant, was admitted to the Pediatric Department of the Queen of the Angels Hospital, April 27, 1953. Mother stated that the patient had had a lump behind the left ear since birth and that it was gradually increasing in size. For the past two weeks the baby had been restless and was not taking food well.

Past History: Full term, normal delivery, normal weight until two weeks previously. *Family History:* Unimportant.

Examination: Showed no pathology in heart or lungs, large swelling in region of left mastoid. Neither membrana tympani could be visualized. White blood count, 19,000. Temperature, 101°F.

Impression: Bony tumor left parietal bone. Seen by the Department of Otolaryngology, April 29, 1953, marked swelling over left mastoid area, softness in superior portion. Right ear normal, left membrana tympani red, with tiny central perforation.

X-ray Examination: Showed soft tissue swelling, no fracture of the skull and no bony involvement. Aspiration into soft area in superior portion of mastoid gave only blood.

Culture: Showed no growth. In view of the leucocytosis, the history, the appearance of the membrana tympani and the condition of the patient, it was decided to perform an exploratory mastoidectomy. This was done April 30, 1953.

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Soft tissues were found edematous. On removing the cortex, pure blood was encountered. Most of the mastoid cells were gone. Mastoid antrum was located. Wound was closed and a small rubber drain left in antrum.

Post-operative course was uneventful. The drain was removed on the third day; culture after 72 hours showed nonhemolytic-staphylococcus aureus; no recurrence of the tumor, and the growth and development of the child has been normal. The perforation in the left membrana tympani closed.

Comment: Whether or not this was a true case of mastoiditis it is difficult to say. Among other conditions considered was a hematoma due to birth injury, but a normal delivery, without the use of instruments, and the fact that the blood was under the cortex, seemed to rule out such a probability. A diagnosis of hemangioma in such a young infant seemed improbable. With progress some involvement of the middle ear and mastoid now seems indicated.

The Alexander Graham Bell Association for the Deaf (formerly The Volta Speech Association for the Deaf) will hold its 1954 Summer meeting in St. Louis, June 14-18 inclusive. The theme of this meeting is "Let's Face the Issues," and some of the questions to be discussed are: "Where should the deaf child receive his education?" "What are the possibilities and limitations of auditory training?" "What are the principal issues in teaching speech?" and "What contributions can be made to the understanding of the deaf child by persons in peripheral fields?"

All sessions will be held at the Chase Hotel, but there will be Open House at Central Institute for the Deaf and St. Joseph Institute for the Deaf.

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IMPROVED MODIFICATION OF RIGHT ANGLE SAW FOR RHINOPLASTY.*

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The right angle saw commonly used in narrowing the bridge of the nose, on which a modification is to be described, includes a heavy handle (Lawle's handle), molded to fit the gripping hand with thumb upward, in order to favor the pressure necessary for cutting bone. This saw was designed to aid in fracturing the anterior maxillary processes; therefore, a strong handle is needed.

The overall length of the saw handle is 4½ in. (10.5 cm.), tapering irregularly toward what represents the lower end, when it is gripped, and is formed to turn at about a right angle and extend forward 1¼ in. (2.8 cm.) to the point where the shank of the saw is fixed. The overall length of the saw arm is 3⅛ in. (7.8 cm.), made of stainless steel. The shank of the saw, which is fastened directly into the handle, is bent laterally at an obtuse angle, which brings the saw edge roughly ¾ in. (1 cm.) to the right of the shank by which it is connected with the handle. At the point of the bend of the saw shank, the toothed portion is dropped downward about ⅛ in. (4 mm.) but remains parallel to the shank. The saw itself is 1½ in. (3.8 cm.) long, finely toothed in the ordinary crosscut manner, with the sides of the points equal.

The modification relates to three particulars:

a. The modified handle is made about ½ in. (1.3 cm.) shorter than the usual saw, and the saw shank is fastened directly into the lower end of the handle, extending in the same general direction. An important part of the modification is that, instead of a right angle being formed between the handle and the saw, a moderate acuteness of this angle is produced by having the saw shank set directly in the base

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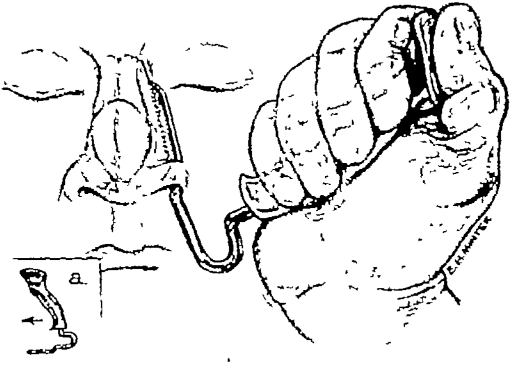
of the handle and angulating the shank, instead of the lower part of the handle itself (see Fig. 1-a). This change of the angle between the handle and the saw increases the potential pressure of the saw upon the bone to be cut.



Proper position of Improved Right Angle Saw.

b. About ⅜ in. (1 cm.) from its insertion into the end of the handle, the shank of the saw is bent to form a U (see Fig. 1-b), which measures ⅝ in. (1.7 cm.) at its open extremity and is ½ inch (1.3 cm.) in depth. This is significant, because it makes it possible for the saw to be easily inserted well up toward the inner canthus, by allowing the overlying tissues to lie in the U-curve, instead of being displaced by upward pressure such as is the case with the other saw.

c. The third detail of the modification is immediately concerned with the cutting edge of the saw, which is of the same size as the original saw, but is slightly thicker. Also, instead of all of the teeth being cut with equal sides, the first five at the tip of the saw are cut so that they point forward, one side of each tooth being longer than the other (see Fig. 1-c), so that when the to-and-fro is made, the effect of the saw is increased on the bony surface of the anterior maxillary process in the region of the inner canthus. In this way the operation is made easier and a more exact and complete fracture can be effected.

To use the saw, an incision is made with a No. 11 Bard-Parker knife at the pyriform crest, just within the naris, and the tissues are undermined with a periosteal elevator. The undermining here should be extensive enough to allow the entrance of the modified saw and its penetration upward to the inner canthus.

Although the electric saw may in general be more useful, there are times when it may not be available; some choose handpower, either by preference or because they are not sufficiently familiar with the electric saw to use it successfully. Such operators will find this modification more fitting and more suited to their purpose.

2104 Spruce Street.

POSTGRADUATE COURSE IN OTOLARYNGOLOGY

The Department of Postgraduate Medicine of the University of Michigan Medical School announces the Otolaryngology Conference to be given at the University Hospital, Ann Arbor, Michigan, April 15-17, under the direction of Dr. A. C. Furstenberg, Chairman of the Department of Otolaryngology, University of Michigan Medical School.

Guest Lecturers: Dr. J. A. Hilger, St. Paul, Minn.; Dr. H. P. Schenck, Philadelphia; Dr. Paul Holinger, Chicago; Dr. K. M. Day, Pittsburgh; and Dr. Lawrence R. Boies, Minneapolis, Minn.

Resident Lecturers: Dr. A. C. Furstenberg, Dr. J. H. Maxwell, Dr. J. E. Magielski, Dr. Thomas Francis, Dr. Jerome Conn, Dr. E. R. Harrell, Jr., Dr. R. B. Sweet, Dr. H. E. Sloan, Jr., and Dr. Irving Blatt.

For further information, address: Dr. H. H. Cummings, Chairman, Department of Postgraduate Medicine, University Hospital, Ann Arbor, Michigan.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

The Sixth International Congress of Otolaryngology will be held in the United States of America in the Summer of 1957. The exact dates and the place of meeting are, at this writing, still undecided. It will be the first International Otolaryngological Congress to be held outside Europe and will afford a welcome opportunity to American otolaryngologists to meet and exchange views with their colleagues from all over the world.

The first of the present series of Congresses met in Copenhagen in 1928 under the presidency of Prof. Schmiegelow; it was attended by 600 members representing 41 countries. The second was held four years later in Madrid, under the presidency of Prof. Tapia; the third in Berlin in 1936, under the presidency of Prof. von Eicken.

At the meeting in Berlin, Holland was designated to take the Congress in 1940 and England, tentatively, in 1944; however, the war intervened, the Berlin records were lost, and further plans remained in abeyance until 1947. At that time, the Dutch committee having indicated its temporary inability to undertake a meeting, the British Association of Otolaryngologists agreed to be hosts to the Fourth Congress, which took place in London in 1949 under the presidency of Mr. V. E. Negus, and was attended by some 1300 members from 45 countries. The Dutch then resumed their obligation and the Fifth Congress was held in Amsterdam, in June 1953, Prof. Eelco Huizinga presiding. The Sixth, to be held in this country in 1957, will be under the presidency of Dr. Arthur Proetz.

Three official themes usually dominate the scientific programs; while the presentations under these heads are by invited speakers, any member of the Congress is eligible to present a paper on a subject of his own choice, and there are sectional meetings in order to accommodate all of the speakers within the time available.

The Congresses have been extremely successful; scientific contributions are many and varied; new friendships are established and old friendships renewed. They have established a sound basis of mutual esteem and understanding by bringing together representatives of countries throughout the world for personal discussion of mutual problems.

Aside from the scientific sessions, social and cultural programs have been arranged which have included specially conducted tours to points of interest in the host city and country. Following some of the Congresses, arrangements have been made for visits to other parts of the host countries for additional special programs. These were both scientific and social in character, and were very successful additions to the Congresses.

PAUL H. HOLINGER, M.D., Secretary-General.

Dr. Sam H. Sanders, Jr., has been named head of the Department of Otology, Laryngology, and Rhinology at the University of Tennessee College of Medicine, succeeding Dr. Charles Blassingame, who asked to be relieved of his administration duties.

Besides his membership in the local and state societies, he is a Fellow of the American Academy of Ophthalmology and Otolaryngology; The American Laryngological, Rhinological and Otological Society; The International Broncho-Esophagological Society; The American Society of Ophthalmologic and Otolaryngologic Allergy, as well as The American College of Allergists; The American Academy of Allergists, and the International Association of Allergists.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

March 1, 1954.

Acousticon Models A-17, A-180 and A-185.
Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 1, New York.

Auditone Models 11 and 15.
Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67 and 70.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac Model M; Mono-Pac Model "Lyric"; Mono-Pac Model "Rhapsody."
Manufacturer: Beltone Hearing Aid Co., 2900 West 36th St., Chicago 32, Ill.

Cleartone Model 500; Model 700; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Model D-1; Dahlberg Junior Model D-2; Dahlberg Model D-3 Tru-Sonic; Dahlberg Model D-4 Tru-Sonic.
Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Fortiphone Models 19-LR; 20A; 21-C and 22.
Manufacturer: Fortiphone Limited, Fortiphone House, 247 Regent St., London W. 1, England.
Distributor: Anton Heilman, 75 Madison Ave., New York 16, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.
Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.
Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn

Maico Model J; Maico Top Secret Model L; Maico Maxitone.

Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis, Minn.

Micronic Model 303; Micronic Model "Mercury"; Micronic Star Model.

Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Classic Model T9; Microtone Model T10; Microtone Model T612.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 106 So. 7th St., Philadelphia 6, Pa.

Normatone Model C and Model D-53.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.

Distributor: Normatone Hearing Aid Co., 22 East 7th St., St. Paul (1), Minn.

Otarion Models B-15 and B-30; Otarion Models F-1, and F-3; Otarion Model G-3; Otarion Model H-1; Custom "5."

Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40. Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny-Mite); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Model 62 Starlet; Model 72; Model 82 (Zephyr).

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Distributor: Radioear Corp., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Silvertone Model H-16, J-92; Silvertone Model P-15.

Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.

Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.
Manufacturer: Solo-Pak Electronics Corp, Linden St., Reading, Mass.

Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966; Sonotone Model 977; Sonotone Model 988.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

Televox Model E.
Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 953; Telex Model 1700.
Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.
Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass

Tonemaster; Model Cameo.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill

Unex *Midget Model 95*; Unex *Midget Model 110*; Unex Models 200 and 230.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal; Zenith "Regent."
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices have vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TRANSISTOR HEARING AIDS ACCEPTED.

Acousticon Model A300; 1 transistor, 2 tubes. Model A-310; 1 transistor, and 2 tubes.

Manufacturer: Dictograph Products., Inc., 95-25 149th St., Jamaica 35, N. Y.

Audivox, Model 71; 3 transistors.

Manufacturer: Audivox, Inc., 123 Worcester St., Boston 18, Mass.

Maico Transist-Ear, Model O; 3 transistors.

Manufacturer: The Maico Company, Inc., 21 N. 3rd St., Minneapolis, 1.

Otarion Model C-15; 1 transistor, 2 tubes.

Manufacturer: Otarion, Inc., 4757 N. Ravenswood Ave., Chicago 40, Ill.

Sonotone Model 1010; 1 transistor, 2 tubes.

Manufacturer: Sonotone Corporation, Elmsford, N. Y.

Telex Model 954; 1 transistor, 2 tubes, and 2 batteries (A & B).

Manufacturer: Telex, Inc., Telex Park, St. Paul, 1.

Zenith Model Royal-T; 3 transistors. Zenith Model Super Royal-T; 3 transistors.

Manufacturer: Zenith Radio Corp., 5801 W. Dickens Ave., Chicago 39, Illinois.

SEMI PORTABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).

Manufacturer: A. M. Brooks Co., 1222 W. Washington Blvd., Los Angeles 7, Calif.

Aurex (Semi-Portable).

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.

Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. Frederick T. Hill, Professional Bldg., Waterville, Me.
Vice-President: Dr. D. E. Staunton Wishart, 170 St. George St., Toronto 5, Ontario, Canada.
Secretary: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Ill.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Statler Hotel, Boston, Mass., May 23-24, 1954.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Gordon F. Harkness, Davenport, Iowa.
First Vice-President: Claude C. Cody, Houston, Tex.
Second Vice-President: Daniel S. Cunning, New York, N. Y.
Secretary: Harry P. Schenck, Philadelphia, Pa.
Treasurer: Fred W. Dixon, Cleveland, Ohio.
Meeting: Statler Hotel, Boston, Mass., afternoon, May 27; all day, May 28.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. LeRoy A. Schall, 243 Charles St., Boston, Mass
President-Elect: Dr. Kenneth M. Day, 121 University Pl., Pittsburgh, Pa.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Meeting: Statler Hotel, Boston, Mass., May 25-27, 1954. (Mornings only.)

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. Dean Lierle, Iowa City, Iowa.
Vice-Chairman: Dr. Fred W. Dixon, Rose Bldg., Cleveland, Ohio.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3, Tenn.
Meeting: San Francisco, Calif., June 21-25, 1954.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Walter H. Theobald, 307 N. Michigan Ave., Chicago 11, Ill.
President-Elect: Dr. Algernon B. Reese, 73 East 71st St., New York 21, N. Y.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Waldorf-Astoria, New York City, Sept. 19-24, 1954.

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Statler Hotel, Boston, Mass., May 17-22, 1954.
Waldorf-Astoria, New York City, Sept., 1954.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Edwin N. Broyles, 1100 No. Charles St., Baltimore 1, Md.
Secretary: Dr. F. Johnson Putney, 255 So. 17th St., Philadelphia (3) Pa.
Meeting: Statler Hotel, Boston, Mass. (Afternoons) May 25-26, 1954.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. Victor Alfaro.
Vice-Chairman: Dr. Irvin Feldman.
Secretary: Dr. Frasier Williams.
Treasurer: Dr. John Louzan.
Meetings are held on the third Tuesday of October, November, March and May, 7:00 P.M.
Place: Army and Navy Club, Washington, D. C.

THE LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. W. L. Hughes, Lamar Life Bldg., Jackson, Miss.
Vice-President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

OTOSCLEROSIS STUDY GROUP.

President: Theo. E. Walsh, 640 So. Kingshighway, St. Louis 10, Mo.
Secretary: Dr. Lawrence R. Boies, Med. Arts Bldg., Minneapolis 2, Minn.
Meeting: Waldorf-Astoria, New York City, Sept., 1954.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. Albert D. Ruedemann, 1633 David Whitney Bldg., Detroit 26, Mich.
President-Elect: Dr. F. Lambert McGannon, 14900 Detroit Ave., Lakewood 9, Ohio.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2, N. Y.
Meeting: Waldorf-Astoria, New York City, September, 1954.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. J. M. Tato, Azcuenaga 235, Buenos Aires, Argentina.
Executive Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U. S. A.
Meeting: Fifth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
President: Dr. J. H. Font, Medical Arts Bldg., San Juan, P. R.
Time and Place: 1956, Puerto Rico.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President Dr Norris J Heckel, Chicago, Ill.
President Elect Dr Arthur S Bristow, Princeton, Mo
Secretary Treasurer Dr Harold Swanberg, Quincy, Ill.
Assistant Secretary Treasurer Dr Jacob E. Reisch, Springfield, Ill
Meeting Chicago, Ill., Sept. 22-24, 1954

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Secretary of Section on Ophthalmology Wendell C Irvine, M.D
Chairman of Section on Otolaryngology Max E Pohlman, M D
Secretary of Section on Otolaryngology Herschel H. Burston, M D
Place Los Angeles County Medical Association Building 1925 Wilshire Boulevard Los Angeles 57, Calif
Time 6 00 P.M., first Thursday of each month from September to June inclusive—Ophthalmology Section 6 00 P M fourth Monday of each month from September to June inclusive—Otolaryngology Section.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY

President Dr Harry Nievert, 555 Park Ave., New York (21), N Y
Secretary Dr Louis Joel Fleit, 66 Park Ave., New York (16), N Y

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY

President Dr Cecil Swann Asheville N C
Secretary and Treasurer Dr Geo B. Ferguson Durham, N Car
Meeting Joint, with South Carolina Society of Ophthalmology and Otolaryngology, Durham, N C, Nov 4-6, 1954

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President Dr David S Asbill, Columbia, S Car
Vice-President Dr John McLean Greenville S Car
Secretary Treasurer Dr Roderick Macdonald Rock Hill, S Car
Meeting Joint with North Carolina Eye, Ear, Nose and Throat Society, Durham, N C., Nov 4 6, 1954

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President. Dr Leland G Hunnicutt, 98 N Madison Ave., Pasadena, Calif.
Secretary Treasurer Dr John F Tolan, 3419 47th Ave., Seattle (5), Wash.
Meeting Honolulu, 1954

THE RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Chairman: Dr. Isaac H. Jones, 635 S. Westlake, Los Angeles, Calif.
Treasurer: Dr. Pierre Violé, 1930 Wilshire Blvd., Los Angeles, Calif.
Program Chairmen:
Otolaryngology: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.
Ophthalmology: Dr. Harold F. Whalman, 727 W. 7th St., Los Angeles, Calif.

Mid-Winter Clinical Convention annually the last two weeks in January at Los Angeles, Calif.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Chas. C. Grace, 145 King St., St. Augustine, Fla.
President-Elect: Dr. Jos. W. Taylor, 706 Franklin St., Tampa, Fla.
Secretary-Treasurer: Dr. Carl S. McLemore, 1217 Kuhl Ave., Orlando, Fla.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

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Treasurer: Dr. Chevalier L. Jackson.
Secretary: Dr. John J. O'Keefe.
Historian: Dr. Herman B. Cohen.
Executive Committee: Dr. M. Valentine Miller, Dr. Charles E. Towson, Dr. Thomas F. Furlong, Dr. Benjamin H. Shuster, ex-officio.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Vice-Chairman: Dr. K. W. Cosgrove, 111 E. Capitol Ave., Little Rock, Ark.
Secretary: Dr. F. A. Holden, Medical Arts Bldg., Baltimore, Md.
Meeting:

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James K. Stewart, Wheeling, W. Va.
Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. C. Otrich, Belleville, Ill.
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CANADIAN OTOLARYNGOLOGICAL SOCIETY
SOCIETE CANADIENNE D'OTOLARYNGOLOGIE

President: Dr. Robert Black, 401 Medical Arts Bldg., Winnipeg, Manitoba.
Secretary: Dr. W. Ross Wright, 361 Regent St., Fredericton, N. B.
Place: Harrison Hot Springs Spa, Harrison Hot Springs, B. C.
Time: June 13-15, 1954.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Oscar Marchman, Jr., Dallas, Texas.
Secretary-Treasurer: Dr. Morris F. Waldman, Dallas, Texas.

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDICO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Salvador Mixco Pinto.
Secretary: Dr. Daniel Alfredo Alfaro.
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MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

President: Dr. Cesar LaBolde, Mexico, D. F.
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Secretary: Dr. Juan de Dios Peza, Mexico, D. F.

FEDERACION ARGENTINA, DE SOCIEDADES DE OTORRINOLARINGOLOGIA.

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Sub-Secretario del Exterior: Dr. Oreste E. Bergaglio.
Secretario del Interior: Dr. Eduardo Casterán.
Sub-Secretario del Interior: Dr.Atilio Viale del Carril.
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ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

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Vice-Presdente: Dr. Luis Suñe Medan.
Secretario: Dr. Jorge Perelló, 319 Provenza, Barcelona.
Vice-Secretario: Dr. A. Pinart.
Vocal: Dr. J. M. Ferrando.

SOCIEDAD NACIONAL DE CIRUGIA OF CUBA.

Presidente: Dr. Reinaldo de Villiers.
Vicepresidente: Dr. César Cabrera Calderin.
Secretario: Dr. José Xirau.
Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Andre Soulas, Paris, France.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa. U. S. A.
Meeting: 3rd International Congress of Broncho-Esophagology.
Time and Place: September or October, 1954, Lisbon, Portugal.

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER—CAMPINAS.

President: Dr. Heitor Nascimento.
First Secretary: Dr. Roberto Barbosa.
Second Secretary: Dr. Roberto Franco do Amaral.
Librarian-Treasurer: Dr. Leoncio de Souza Queiroz.
Editors for the Archives of the Society: Dr. Guedes de Melo Filho, Dr. Penido Burnier and Dr. Gabriel Porto.

SOCIEDAD CUBANA DE OTO-LARINGOLOGIA.

President: Dr. Reinaldo de Villiers.
Vice-President: Dr. Jorge de Cárdenas.
Secretary: Dr. Pablo Hernandez.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA.

Presidente: Dr. Aldo Remorino.
Vice-Presidente: Dr. Luis E. Olsen.
Secretario: Dr. Eugenio Romero Díaz.
Tesorero: Dr. Juan Manuel Pradales.
Vocales: Dr. Osvaldo Suárez, Dr. Nondier Asís R., Dr. Jorge Bergallo Yofre.

BUENOS AIRES CLUB OTORINOLARINGOLOGICO.

Presidente: Dr. K. Segre.
Vice-Presidente: Dr. A. P. Belou.
Secretario: Dr. S. A. Aranz.
Pro-Secretario: Dr. J. M. Tato.
Tesorero: Dr. F. Games.
Pro-Tesorero: Dr. J. A. Bello.

SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLOMBIA).

Presidente: Dr. Alfonso Tribin P.
Secretario: Dr. Felix E. Lozano.
Tesorero: Dr. Mario Arenas A.

SOCIEDAD ESPANOLA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. D. Adolfo Hinojar Pons.
Vice-Presidente: Dr. D. Jose Perez Mateos.
Secretario General: Dr. D. Francisco Marañés.
Tesorero: Dr. D. Ernesto Alonso Ferrer.

ASOCIACION DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DE GUATEMALA

Presidente: Dr. Julio Quevedo, 15 Calle Oriente No. 5.
First Vice-Presidente: Dr. Héctor Cruz, 3a Avenida Sur No. 72.
Second Vice-Presidente: Dr. José Luis Escamilla, 5a Calle Poniente No. 48.
Secretario-Tesorero: Dr. Horace Polanco, 13 Calle Poniente No. 9-D.

FIRST CENTRAL AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY.

President: Dr. Victor M. Noubleau, San Salvador.
Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

SOCIEDAD DE ESTUDIOS CLINICOS DE LA HABANA

Presidente: Dr. Frank Canosa Lorenzo.
Vice-Presidente: Dr. Julio Sanguily.
Secretario: Dr. Juan Portuondo de Castro.
Tesorero: Dr. Luis Ortega Verdes.

FOURTH LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Dario.
Secretary:
Meeting: Lima, Peru, 1957.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY

President: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis, Mo.
General Secretary: Dr. Paul Holinger, 700 No. Michigan Ave., Chicago (11), Ill.
Meeting: U. S. A., 1957.

THE
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PRESERVATION OF HEARING IN SURGERY FOR CHRONIC EAR DISEASE.*†

A Consideration of Factors Involved.

ARTHUR L. JUERS, M.D.,
Louisville, Ky.

Several excellent articles have appeared in the literature recently concerning the modified radical mastoidectomy (Bondy operation, atticomastoidectomy). The historical aspects of the subject have been reviewed in detail by previous authors[1,2] and will not be considered further here. This discussion will be concerned chiefly with the factors which influence the preservation or improvement of hearing in this type of ear surgery. In order to discuss the problem adequately, a brief review of the genesis of the lesions amenable to this type of surgery will be necessary.[3]

In general, the case suitable for modified radical mastoidectomy has attic cholesteatoma (cholesteatosis) originating from an attic retraction type of perforation without extension into the middle ear. In some instances the cholesteatoma will eventually spread into the mastoid antrum and mastoid cells in the presence of pneumatization; however, if cholesteatoma has invaded the middle ear to the extent that it cannot be completely exteriorized and yet retain an inflatable tympanic cavity, then a complete radical mastoidectomy is indicated.

A clinical classification of cholesteatoma has been presented previously, together with the mode of genesis of the attic retraction type of cholesteatoma.[3] This phase of the cholesteatoma problem will not be discussed again here, but must be fully understood in order that the technical aspects of surgery in these cases may be fully comprehended.

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* Presented at the meeting of the Cincinnati Society of Otolaryngology, November, 1953.

† From the Department of Otolaryngology, University of Louisville.

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The indications for surgery in attic retraction pathology with suspected cholesteatoma will be briefly reviewed. I believe that there is a trend toward earlier surgery in these cases, because in some instances conservative management results in eventual invasion of the labyrinth and the facial canal; furthermore, the current surgical approach permits the retention of serviceable hearing in a high percentage of cases. Most of the complete radical mastoidectomy operations I have performed in recent years have been on cases of long-standing and, in some instances, neglected attic retraction cholesteatomas in which the tympanic cavity had become filled with cholesteatoma extension.

INDICATIONS FOR SURGERY.

1. Small Attic Opening. Reference to Fig. 1 will reveal the danger of the presence of an attic cholesteatoma with a small attic opening. The exfoliated epithelium from the matrix cannot be extruded through the "bottleneck" at the perforation, and expansion of the lesion internally is inevitable. Conservative treatment of such a lesion will at best merely delay the time when a complication will develop; however, if there has been considerable erosion or absorption of the lateral margin of the opening (floor of the attic and antrum), then conservative management may be justifiably considered. If after a short period of treatment the ear does not become dry, then there undoubtedly is extension of disease beyond the reach of local treatment and surgery is indicated.

2. Persistence of Drainage. This has already been mentioned but deserves being listed as a separate indication. In the younger age groups the persistence of drainage is particularly significant as an indication for surgery. Early surgery in such cases gives the best assurance of retaining useful hearing.

3. Extension into Contiguous Structures. The development of facial, labyrinthine or intracranial extension is an unquestioned indication for immediate surgical intervention.

4. X-ray Evidence of Cholesteatoma. Most cases of attic cholesteatoma treated surgically have not revealed preoperatively the characteristic picture of cholesteatomatous erosion. The conventional X-ray views usually show either sclerosis or, if the mastoid is pneumatized, a cloudiness of the cells.

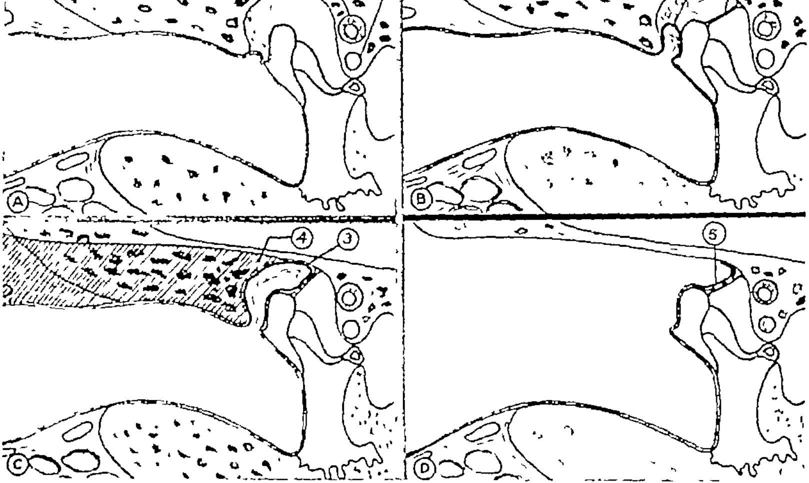

Fig. 1

A, normal middle ear and external meatus.

B, 1. Connective tissue membrane sealing off the attic from the tympanic cavity. The exact position of this membrane varies considerably. 2. Beginning retraction of Shrapnel's membrane into the attic as a result of negative pressure in the attic. Desquamated epithelium is collecting in the invaginated sac.

C, 3. Cholesteatoma has filled the attic as far as the membrane (B, 1). Anterior and posterior to the plane represented in this diagram the cholesteatoma usually lies against the bony labyrinth and not infrequently against the facial nerve as well. In long-standing cases the cholesteatoma may invade the tympanic cavity. 4. The shaded area is the lateral attic-antral bone which is removed in the modified radical mastoidectomy.

D, cholesteatoma and lateral attic-antral bone has been removed. 5. The medial matrix remains. If this matrix is entirely removed the tympanic cavity must be sealed off with a meatal plastic flap or skin graft.

A minority of cases with proven cholesteatoma show a circumscribed area of erosion. When such an area of erosion is noted on routine X-ray views, the lesion is large and, with few exceptions, the case should be treated surgically.

SURGICAL TECHNIQUE.

I prefer the Lempert endaural approach for surgery in cases where the pathology is located chiefly in the attic and antrum. If there is extensive pneumatization, then the postauricular approach might have some advantage. Good illumination and the use of loupe magnification for the final phase of the operation are essential.

The question of the preservation of the matrix is important in considering technique. If the mastoid is pneumatized, I think that it is preferable to remove all of the matrix, because of the likelihood of the presence of fingerlike projections into underlying cells. In sclerosed mastoids I remove all matrix in the periantral and posterior antral area. I elevate the matrix in the medial attic-antral region and, if no finger-like projections are noted, I preserve this portion unless there is surface evidence of ossicular necrosis such as granulations. Complete exteriorization of the antrum and attic is essential—especially the anterior attic area.

I find it increasingly difficult to justify more or less routine preservation of the matrix. The fact that a 20-decibel hearing level after surgery is frequently maintained, even though the incus is absent, causes one to consider more critically the advisability of compromising on the question of the removal of all pathology, because of uncertainty of being able to retain serviceable hearing.

CLINICAL OBSERVATIONS.

The problem of the integrity of the ossicular chain and its relation to the postoperative hearing level can be discussed best in connection with illustrative case reports. Four possibilities concerning the state of the ossicles are listed, and one or two cases are cited as examples of each situation. The audiometric tests on the operated ear only are shown here.

1. Ossicular Chain Intact. Close inspection of the ossicular chain at surgery revealed that the chain was functionally intact in relatively few. Fig. 2 shows the preoperative and post-

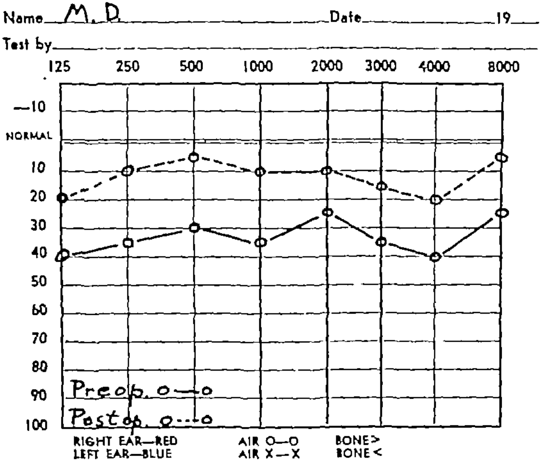
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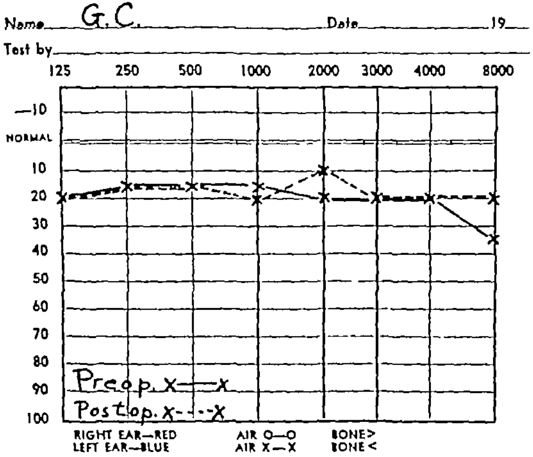
Fig. 2.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Postop. x---x
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LEFT EAR—BLUE
AIR O—O
AIR X—X
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Fig. 3.

operative audiogram of a case in which the ossicular chain appeared to be intact under the matrix; the ossicles were not disturbed and the matrix was left *in situ.* This patient was 10 years of age at the time of surgery. There is a possibility that movement of the ossicles may become impeded later by adhesive changes.

2. Ossicles Destroyed by Disease. I find that in the majority of my cases the continuity of the ossicular chain has been disrupted by attic pathology; however, despite this fact, a serviceable level of hearing is maintained in many. Fig. 3 illustrates such a case. The long process of the incus was absent, and the posterior superior portion of the pars tensa was in contact with the upper promontory and the stapes. Cholesteatoma was extensive, and finger-like projections were found in some cells. All matrix was removed. The remains of the incus and the head of the malleus were removed. The middle ear was sealed off from the attic with a small skin flap rotated from the anterior superior meatal wall. This case illustrates the degree of hearing possible when there is contact between the pars tensa and the stapes, in the absence of a functioning ossicular chain. The tympano-stapedial contact was not disturbed and the hearing after surgery was the same as that before. Inflation revealed an inflatable air-containing space over the round window.

Fig. 4 shows the preoperative and postoperative audiograms on a 20-year-old patient who had a history of bilateral otorrhea since two years of age. In both ears the pars tensa was adherent to the upper three-fourths of the promontory, and the stapes was observed as pushing out the overlying pars tensa in tent-pole fashion. Both ears were operated upon, and the incus was completely gone in each ear. The marked degree of adhesion of the pars tensa to the promontory around the stapes undoubtedly impeded stapedial movement and accounted for the greater hearing loss in this case, as compared to the previous one. Had this patient been operated on earlier in life, a better hearing level might have been preserved. The matrix on the left side was completely removed, because of a projection deep into the petrous pyramid behind the superior semicircular canal.

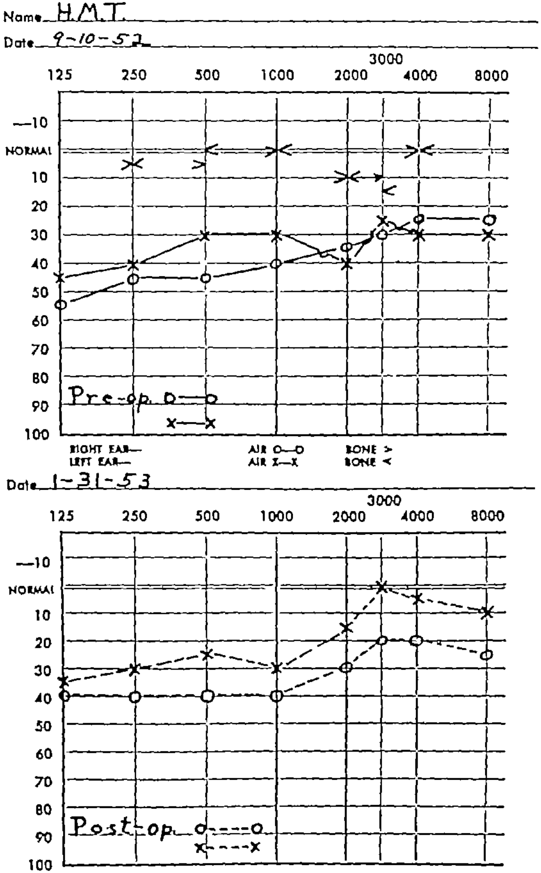
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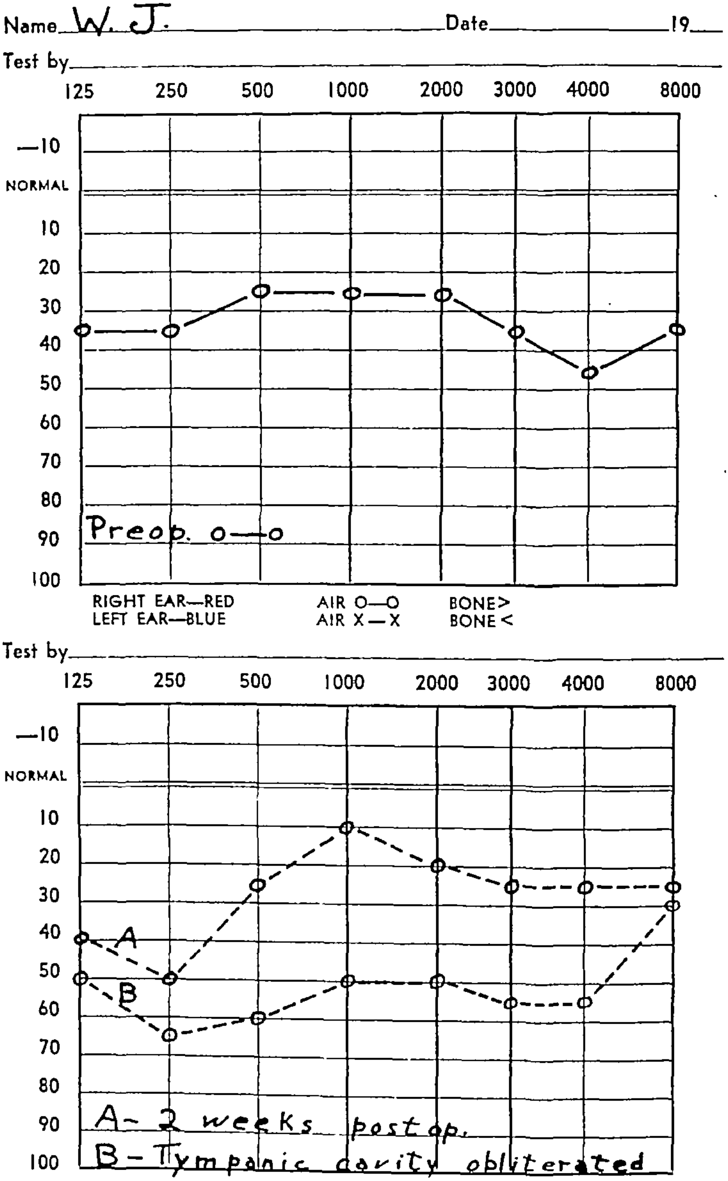
Fig. 4.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A
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A- 2 weeks post op.
B- Tympanic cavity obliterated

Fig. 5.

Fig. 5 shows the audiograms on another patient in whom the long process of the incus was partially destroyed by pathology. After surgery a serviceable level of hearing was preserved; however, a few months later the tympanic cavity became obliterated as a result of the entire pars tensa becoming adherent to the promontory. Inflation could no longer be accomplished, and the hearing dropped to a 50-decibel level. This illustrates that an inflatable air-containing space over the round window is one of the prerequisites to having serviceable hearing after a modified radical mastoidectomy.

3. Ossicular Chain Fixed—Removed at Operation. In one case, the preoperative audiogram indicated a complete conduction deafness (see Fig. 6). The Eustachian tube inflated, and the middle ear was free of fluid. At surgery the incudo-malleolar articulation was found to be ankylosed. The incus and head of the malleus were removed. The hearing after surgery came up to a 20-decibel level.

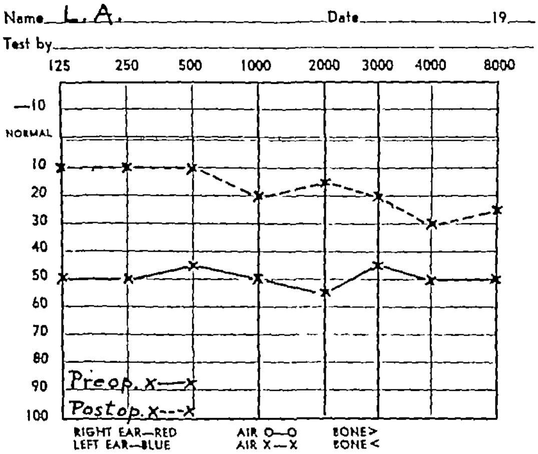

FIG. 6.

4. Ossicular Chain Intact and Functioning—Removal of Incus Necessary to Permit Removal of Pathology. In the past there has at times been a tendency to compromise with complete removal of pathology, for fear of disturbing a well functioning ossicular chain. Conversely, there has been a tendency by others under these circumstances to do a complete radical mastoidectomy, under the erroneous conception that the removal of the incus would produce a 50-decibel loss anyway. Since it is possible to have 20-decibel hearing level without the presence of the incus, neither the compromise nor the unnecessarily radical surgery is justified.

Fig. 7 shows the audiograms on a patient with a recurrent mastoiditis. A simple mastoidectomy had been done two years previously. Following swimming there had been a recurrence of otorrhea and symptoms suggesting serous labyrinthitis. It will be noted that before surgery the bone conduction threshold was somewhat elevated. Preoperatively the air-bone gap is only 20 decibels, indicating fairly good ossicular chain function. A small perforation was present in the pars tensa. At surgery no cholesteatoma was found, but a pyogenic membrane lined the antrum and the previous operative cavity. It was decided to do a modified radical mastoidectomy in order to eliminate any possibility of a recurrence of infection in the antrum in the event that an otitis media recurred. There was some question of osteitis involving the incus and it, together with the head of the malleus, was removed. The middle ear was sealed off from the attic and antrum with a fenestration type of metal flap, as there was no attic perforation. Postoperatively, the hearing did not reach the optimum level until the central perforation healed and the posterior superior quadrant of the pars tensa retracted and apparently established contact with the stapes. It will be noted that the bone conduction acuity improved after the elimination of the infection. The patient stated that the postoperative hearing level was equal to that present before the development of the otitis media.

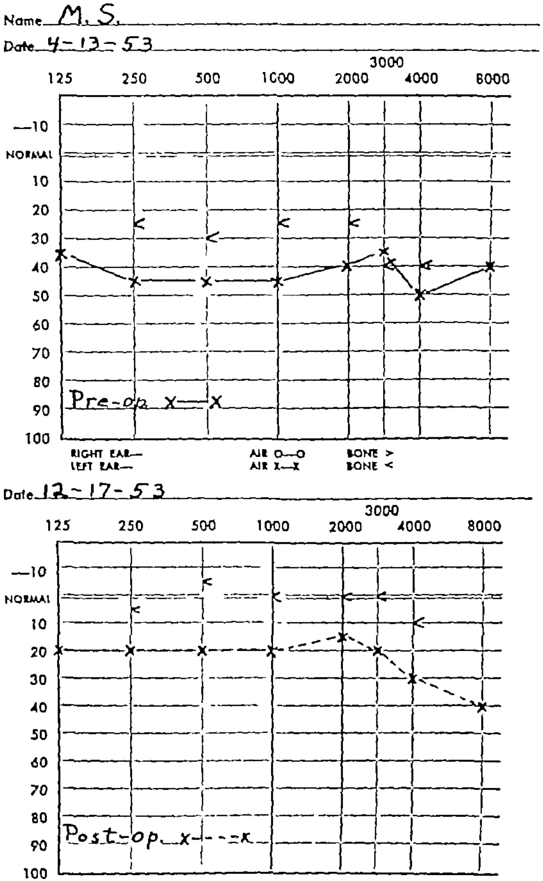
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Fig. 7.

In Fig. 8 are shown the audiograms of a patient in whom the incus and malleus head had to be sacrificed in order to remove cholesteatoma which had extended medial to the ossicles. All matrix was removed. The postoperative audiogram shows an air-bone difference of about 30 decibels instead of the usual 20 noted in most cases in which the incus

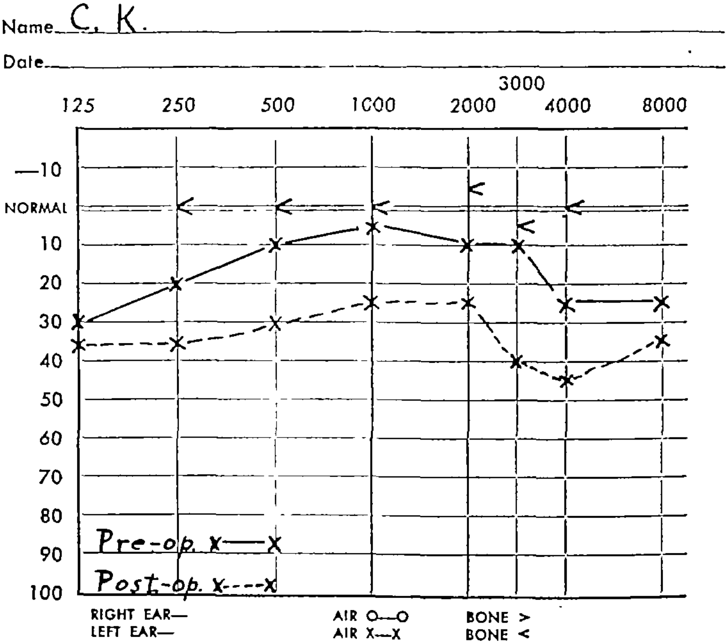

Fig. 8.

was absent. It is my belief that in this case there may have been poor contact between the pars tensa and the stapes after healing took place. In view of this possibility I am suggesting a surgical procedure which I plan to carry out in similar situations in the future to insure contact between the pars tensa and the stapes. Such contact should assure an excellent chance of obtaining a 20-decibel postoperative level if the bone conduction threshold is normal and the mobility of the stapes is unimpeded.

MYRINGOSTAPEDIOPEXY.*

This procedure is suggested when the following situations exist after the pathology has been removed in performing a modified radical mastoidectomy:

1. A functioning incus has been removed for pathological reasons and the pars tensa is not in contact with the stapes.

2. A non-functioning (ankylosed) incus has been removed.

The procedure is illustrated in the four sketches in Fig. 9. The incisions in the meatal flap should not be made until all pathology has been removed, because the flap must be tailored to suit the needs of each individual case. The illustrations indicate a method of incisions when the attic perforation is small and anterior. If the anterior recess of the attic is large, incision No. 3 should be placed more toward the anterior surface of the external meatus, in order to provide sufficient skin to seal off the middle ear completely. If a small attic fistula remains after surgery, the patient will have drainage after each cold and will be dissatisfied with the surgery because his ear still drains. Some of the failures to obtain a dry ear may be due to this fact. The hearing after surgery may not reach its optimum level unless the tympanic cavity is entirely sealed off.

There are two basic steps in carrying out this procedure: first, the posterior margin of the pars tensa together with several mm. of adjacent meatal skin should be mobilized and displaced medially to provide contact between the pars tensa and the stapes. If the chorda tympani prevents suitable contact, it may need to be sacrificed; the second step is to seal off the tympanic cavity from the attic and antrum with a rotated pedicle skin flap as indicated. If the perforation is more posterior in position than indicated in the sketch, the pedicle of the flap may be created antero-superiorly by varying the incisions accordingly. Modifications must be made to suit each individual case. Occasionally both an anterior and posterior flap may be necessary, with approximation of the two flaps in the mid-attic area. I believe that the actual margin of the attic perforation should be excised in order to eliminate any irregular edge of squamous epithelium which might become buried beneath the edge of the flap and be a nidus of a future cholesteatomatous cyst.

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* This term was suggested by Dr. Kurt Tschiassny, Cincinnati, Ohio.

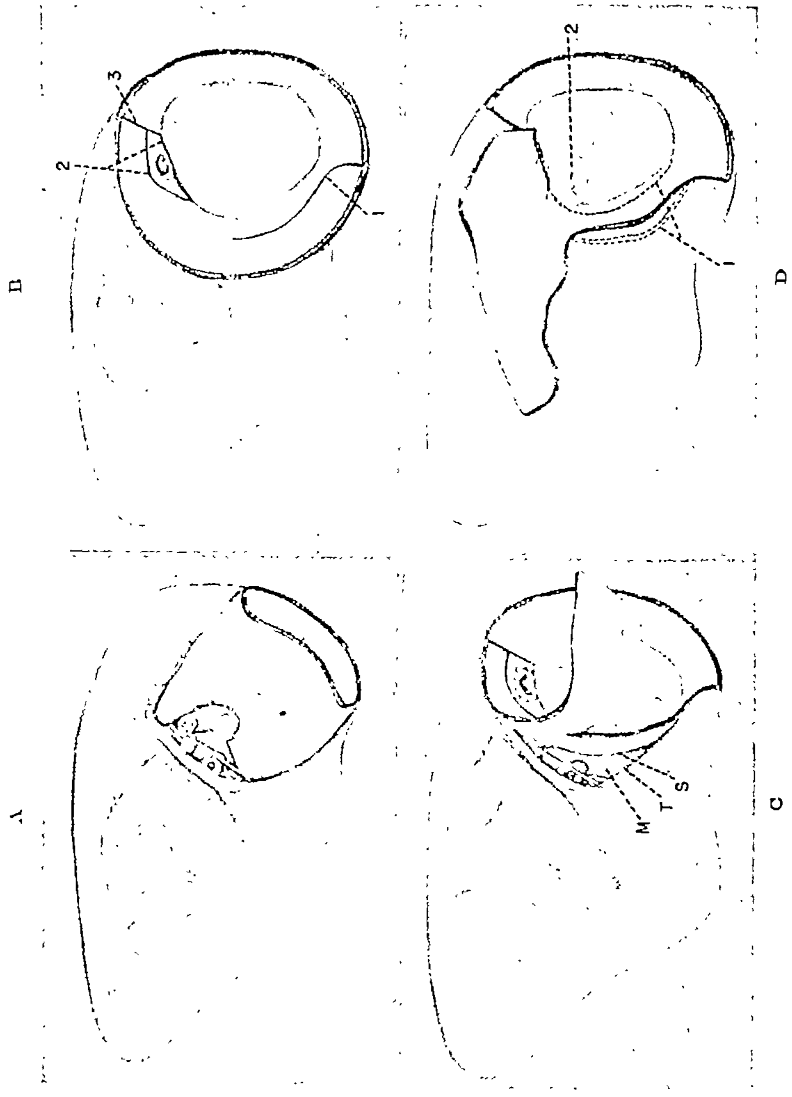

Fig. 9. These are photographs of colored sketches drawn from wax models to represent the four essential steps of myringostapediopexy.

A.—All attic pathology, incus and head of the malleus have been removed. The margin of the original attic perforation is shown at the medial edge of the reflected meatal lining.

B.—Meatal incisions necessary to mobilize flap to permit sealing off of the tympanic cavity from the attic. Incision 3 may be placed more anteriorly if more skin is needed in the anterior attic area. The margin of the perforation is excised by incision 2.

C.—The tympano-meatal flap is freed from the posterior tympanic ring. M.-mucosa on promontory. T.-posterior tympanic ring. S.-posterior edge of tympanic membrane and adjacent meatal skin separated from tympanic ring.

D. Mobilized meatal flap rotated to seal off tympanic cavity. Posterior tympano-meatal flap displaced medially from original position (1) to establish contact between pars tensa and stapes at (2).

If a surgeon prefers, he may elect to use a skin graft to seal off the tympanic cavity instead of a pedicle flap. The disadvantage of using a graft for this purpose is that a failure of the graft to take would result in an attic fistula, and a secondary closure would be necessary if an ideal result is to be obtained.

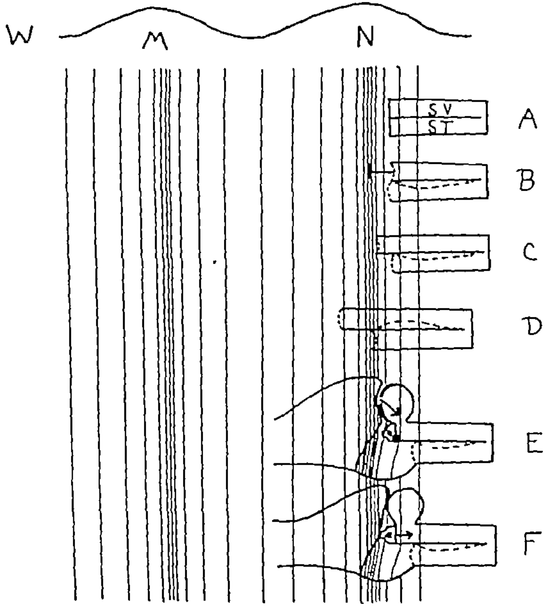

Fig 10 One complete cycle of sound pressure is shown in the usual manner in W. Pressure peaks are at M and N Below are drawn the pressure peaks in a frontal manner Bekesy's box representations of the cochlea have been modified and adapted to show the probable mechanics of the cochlear fluid movement incidental to the application of sound pressure.

A—Pressure at the two cochlear windows is equal and consequently there is no displacement of the basilar membrane

B—A conduction mechanism—represented by the T—against the oval window produces a downward basilar membranes displacement when the pressure peak hits the membrane of the conduction mechanism

C—Difference of sound pressure at the two windows causes displacement of the basilar membrane

D—The same cochlea a moment later with a reversal of pressure relationship

E—Diagrammatic sketch of a fenestrated ear The pressure peak displaces cochlear fluid inward at the fenestra—indicated by arrow.

F—This indicates the relationship established in a myringostapediopexia or in an ear on which the Pattee operation has been performed. The incus is absent and the pars tensa (or skin graft) is in contact with the stapes.

A diagrammatic representation of the probable mechanics of such a reconstructed middle ear is shown in Fig. 10. Davis and Walsh[4] have found that the best air conduction level obtainable after fenestration is 20 decibels below the bone conduction threshold. An analysis of the hearing results obtained in patients on whom a modified radical mastoidectomy has been performed indicates that if the incus is absent and there is contact between an intact pars tensa and the stapes, the air-bone gap (unrestored conductive residue) is also 20 decibels. A similar situation is created by Pattee's operation for congenital atresia. Apparently the ossicular chain adds about 20 decibels to the air conduction hearing acuity.

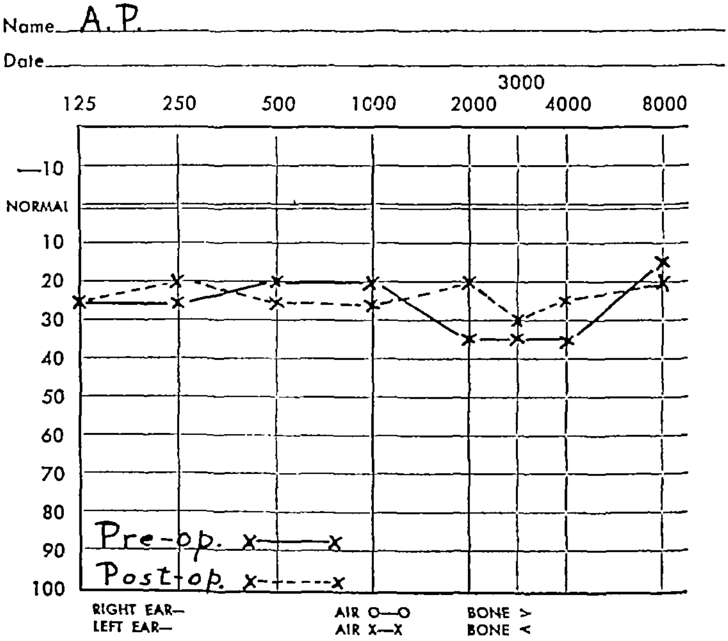

Fig. 11.

Fig. 11 shows the audiograms of a patient on whom a myringostapediopexis was carried out. After removing the attic cholesteatoma some granulations were noted on the head of the malleus. Consequently the incus and malleus head were removed. A serviceable postoperative level of hearing was obtained.

SUMMARY AND CONCLUSIONS.

1. In properly selected cases, a modified radical mastoidectomy (atticomastoidectomy, Bondy mastoidectomy) enables the surgeon to preserve serviceable hearing, and at the same time eliminate pathology originating in the attic.

2. The following prerequisites are essential for a good hearing level after surgery:

- *a.* An air-containing inflatable space over the round window.
- *b.* An intact pars tensa.
- *c.* No fistula from the tympanic cavity into the attic.
- *d.* An intact, or unimpeded ossicular chain

or

if the incus is removed, contact between the pars tensa and the stapes should be assured. If such contact exists before surgery this should not be disturbed.

If such contact is not present, it should be established by the surgical procedure described, *i.e.*, myringostapediopexy.

3. If there is considerable conduction deafness before surgery, the incus and malleus head should be removed and myringostapediopexy accomplished. If serviceable hearing is not restored after complete healing takes place, a secondary fenestration might be considered at a later date if the need for serviceable hearing in the ear in question is sufficient.

4. Each case is an individual problem, and the surgical management is dictated by a thorough study of the hearing before surgery and careful observation of the pathology and status of the ossicular chain during surgery.

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RECENT TRENDS IN THE MANAGEMENT OF CANCER OF THE MOUTH.*†

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(By Invitation).

Louisville, Ky.

Cancer of the mouth may, for convenience, be discussed under three distinct areas: the lip; the tongue, floor of the mouth and gingivae; and the tonsil and oropharynx. Of these, cancer of the lip is the most common and the least dangerous of all. It tends to be of a relatively low grade of malignancy, metastasizes relatively late and then, as a rule, only within the cervical lymph node areas. It has been stated on good authority that not more than 10 per cent of the cancers of the lip will ever metastasize beyond the neck, even in untreated and fatal cases.

In years past it was commonly said that the small primary cancer of the lip could be treated equally well by irradiation, by surgical excision, or by local destruction; the latter, a type of therapy formerly much in vogue among dermatologists. Today we can still say with complete justification that either irradiation or surgery will cure the small and early lesion. Though our radiologic friends may not agree, it is my firm conviction that the larger and more bulky lesions can be excised and immediate repair carried out with less ultimate scarring than is the case if they are destroyed by irradiation. One cannot eradicate malignant tissue by any method without a need for replacement of such tissue, if the lesion be large. I personally prefer to carry out reconstruction at once, with normal tissue, rather than following irradiation, though recognizing full well that modern radiotherapeutic techniques are vastly more effective and less destructive to normal tissue than was the case only a few years ago.

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So-called "prophylactic neck gland dissection" which was ardently advocated, in fact even insisted upon by many a few years ago, has now largely given way to the more conservative point of view that if the patient can be seen at frequent intervals it is safe to withhold neck dissection unless or until the appearance of palpable nodes indicates that it will be necessary. A considerable portion of these cases will be cured without ever requiring neck surgery.

A word should be said at this point about our present concept of what constitutes an adequate neck node dissection. Formerly, it was a common practice to leave the jugular vein and the sterno-mastoid muscle intact while attempting to remove the cervical lymph nodes surrounding these structures. It is now generally accepted that an adequate block resection of the cervical nodes can be carried out only if these structures, and perhaps other important ones which might be involved, are also removed. One should not hesitate to remove whatever structures are necessary to obtain adequate excision of all involved nodes. ✓

For example, the former viewpoint that sacrifice of both jugular veins in bilateral neck node dissections might be hazardous, has now given way before the weight of evidence of many patients who have had simultaneous radical resections on both sides of the neck at one or two sittings without serious physiologic disturbance. Likewise, we no longer feel that the presence of carcinoma in two noncontiguous triangles of the neck represents a contraindication to radical neck node dissection. The entire problem of neck dissection and factors which influence it has been excellently stated by Hayes Martin and his associates.[1] Probably better than anything else, this paper presents the viewpoint which a large number of us hold today, that neck node dissection need not be done in all cases of lip cancer but in only those where palpable lymph nodes develop, but that where such dissection is to be done it must be a more radical procedure than has been the custom in the past.

Cancer of the Tongue and Floor of the Mouth: This is one of the most malignant of all the mouth carcinomas with which we have to deal, and it is the one in which metastasis to the cervical lymph node areas occurs most directly and most early. Surely it is one of the areas in which experience with past programs of treatment has been most disappointing. Some idea of the change in approach to the treatment of cancer in this area is apparent from the statement made in 1859, just short of 100 years ago, by Samuel D. Gross, one of the ablest surgeons of his day, in the second edition of his "System of Surgery":

> "The treatment of this affection (carcinoma of the tongue) has generally, at least until lately, been by ablation, either by the knife or ligature. The utility of such proceeding, however, admits of great doubt, and my own opinion has long been that the less we interfere with the disease in this way the better. The results of experience are certainly corroborative of this conclusion . . . . . . With judicious management, it is astonishing how long, in many cases, the disease may be kept in abeyance and the final issue warded off"[2].

It appears quite obvious that Dr. Gross must have been sadly discouraged by results of most of the excisional types of therapy which had been followed up to this point; however, it was not long before more radical attempts were made to cure oral cancer and before the modern approach to the so-called "cancer field surgery," including both primary and metastatic nodes in one area appeared. Kocher, in 1880, reported resection of the tongue for cancer, carried out through the submaxillary triangle, together with removal of cervical lymphatic and salivary gland tissue. He later proposed that it might be worthwhile regularly to remove cervical lymph nodes. In 1894, Halsted[3] reported a considerable series of radical mastectomies, emphasizing the en bloc removal of the primary lesion, together with all adjacent lymph node areas. Miles[4] in 1908 described a similar technique for resection of carcinoma of the rectum. Despite general acceptance of the importance of such resection of carcinoma and nodes in continuity, until recently there was a persistent tendency to ignore this principle in treating oral cancer and to deal with the primary mouth lesion, and the lymph nodes to which it might be tributary, as separate and distinct problems.

Increasing perfection of irradiation therapy during the third and fourth decades of this century led many to think in terms of irradiation for the primary lesion and, if it were controlled, later excision of the cervical lymph nodes. Irradiation was also attempted for the neck areas, but there has been increasingly wide acceptance of the fact that this is rarely a successful means for eradicating carcinoma in the cervical nodes, while a properly performed radical lymph node dissection may be expected commonly to accomplish this purpose; however, even then, there remains an area between the primary lesion and the neck which is not reached by either method, and which is a potential source for subsequent development of metastatic cancer. A dramatic demonstration of this deficiency of the "two-stage" manner of dealing with these lesions was seen in a tumor clinic which I attended elsewhere about three years ago.

A patient with a small proven primary carcinoma of the tongue underwent an adequate hemiglossectomy. He subsequently had a complete radical neck node dissection and none of the nodes showed carcinoma. The man failed to keep in touch, but returned something over a year later with a large, fixed, deeply infiltrated carcinoma lying within the floor of the mouth in the area between the sites of resection of tongue and nodes. It is seldom justifiable to draw conclusions on the basis of one case, but I can think of no more dramatic argument in favor of a resection of carcinoma and nodes in continuity, at one sitting, than the demonstration of what happened to this patient in whom lymphatic tissue was allowed to remain between the primary and the neck lymphatics, both of which were adequately excised.

Increasing dissatisfaction with irradiation therapy for the cervical areas and difficulty with later resection in these areas due to deep fibrosis and scarring, has led many to feel that radiation should not be used in treating the cervical nodes. Concomitantly with this, we find the scope of surgery increasing in all areas of the body. A better understanding of the physiologic needs of the patient, tremendous advances in anesthesia and a wider use of blood have all extended operative indications tremendously in the last 10 or 15 years. The advent of antibiotic therapy has decreased the hazard of infection, which had been one of the chief difficulties with radical surgery.

These factors, together with newer techniques for resection and reconstruction, have made it possible as never before, to apply the accepted principles of cancer surgery to cancer of the mouth. Radical resection of the primary lesion in continuity with its tributary lymphatic areas has thus developed as a wholesome trend in the present day approach to carcinoma of the mouth.

Techniques for this procedure are well recognized and have been adequately described by several authorities.[6,7,8] As a general rule proximity of these lesions to the mandible will require resection of varying amounts of the mandible itself, including the periosteum. There are occasional cases in which it is justifiable to leave the mandible if it is possible to do so with at least a one-centimeter margin between it and the edge of the lesion. Under these circumstances it should be remembered that there is good experimental evidence that the lymphatics connecting the tongue and floor of the mouth with the neck, pass through the mandibular periosteum; thus even though one resorts to the "pull-through" type of operation, it is important to include the periosteum of the mandible in the resection.

As a general rule, it is wise not to lean too far on the side of conservatism but to resect a segment of the mandible whenever there is any question about the wisdom of the less radical procedure. The described operation of resection in continuity has so far shown very promising results with a higher proportion of cure rates than the older procedure of treating the tongue and the lymph node areas as two separate problems ignoring the tissues between them.

Carcinoma of the tonsil and oro-pharynx, the third type to be considered in this discussion, is probably one of the most unsatisfactory problems in all cancer treatment. The area is inaccessible; the lesions are apt to be of a high degree of malignancy, and their inaccessibility makes them difficult for treatment by either irradiation or resection. Because of the great technical difficulty in obtaining a wide excision of these lesions, there are many who feel that most of them are better handled by irradiation, followed thereafter by radical lymph node dissection in the neck. The answer to the best treatment of this cancer, so far as I am concerned, is still not at hand. It is my own personal feeling, and I believe that of the tumor clinic with which I am most frequently associated, that most of these lesions are better handled in the manner I have described; that is, by irradiation of the primary lesion and radical resection of the cervical lymph nodes.

One should bear in mind, however, that the same type of approach which I have described for resection of the tongue, mandible and neck nodes, can also be extended posteriorly to include the oro-pharynx and tonsillar areas without too great mutilation of the patient Adequate excision requires a more formidable and a more mutilating operation than the described *en bloc* resection for cancer of the tongue and floor of the mouth. This is, however, sometimes justifiable in late or extensive lesions, or in those which have proven resistant to radiation

A short film from the University of Louisville teaching files will illustrate better than any words of mine, the type of operation which appears to offer most hope to the patient with oral cancer

The patient also illustrates the problem which has recently received some attention under the term "multi-centric origin" of cancer, or "field cancer."

CASE REPORT

A G a colored male, age 47, was first seen in November 1949 at which time biopsy revealed a small squamous cell carcinoma of the tongue to be present in an ulcer which was 3 x1½ centimeters in size in the anterior portion of the tongue adjacent to the floor of the mouth A hemiglossectomy, hemimandibulectomy, and radical neck node dissection in continuity were carried out. (A tracheotomy was performed as part of this procedure) He was discharged from the hospital in December 1949 The patient remained well for two years and was repeatedly observed in the tumor clinic during that period He was readmitted in April 1952 at which time he had a small lesion at the base of the tongue on the left side which proved to be an infiltrating carcinoma This was treated by X ray therapy since it was a small area quite posteriorly situated and one which it was felt would not require a second radical procedure like the one done on the right.

In March 1953 there was a small area of thickening at the anterior portion of the tongue, biopsy revealed this to be an *in situ* carcinoma which had been excised for biopsy The patient was readmitted in April 1953 At that time he gave no evidence of any recurrence anywhere in his mouth or neck, however he was having difficulty in swallowing and an esophagoscopy revealed a squamous cell carcinoma of the esophagus histologically Grade II An exploratory thoracotomy was carried out and the carcinoma of the esophagus was found to involve the trachea and to be non resectable A gastrostomy was carried out, but the patient died of carcinoma about two months thereafter Autopsy revealed the carcinoma of the upper end of the esophagus which had perforated into the trachea together with another ulcerative lesion at the lower end of the esophagus which showed *in situ* carcinoma

In summary, then, this patient had three separate infiltrating carcinomas, all believed by the pathologist to be primary: in 1949 of the right tongue, in 1952 the left tongue adjacent to the tonsilar fossa, and in 1953 at the upper end of the esophagus. He also had two other areas from which diagnoses of *in situ* carcinomas were returned, and which were also felt to be independent in origin: in 1953 the left tongue, anteriorly; in 1953 the esophagus, 2½ centimeters from the cardia. Thus, this patient, though cured of his first carcinoma by a radical resection, subsequently died of an independent carcinoma of the esophagus.

CONCLUSIONS.

1. Lip cancer may be eradicated by irradiation or surgical excision. If the patient develops palpable cervical lymph nodes, a truly radical cervical node dissection will be required on one or both sides.

2. Most cancer of the tongue and floor of the mouth is best handled by a resection of the primary lesion in continuity with a radical neck node dissection.

3. Carcinoma of the tonsil and oro-pharynx is a problem not yet solved. At present we favor irradiation of the primary followed by radical cervical node dissection extending up to the irradiated area. Occasional cases may require wide resection including the mandible, floor of the mouth and cervical node areas, an extension of the operation described for tongue and mouth cancers.

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SMOKER'S LARYNX.[*][†]

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Entertainment today is being provided by television and radio to an ever increasing degree. Cigarette companies are among the biggest sponsors. Smoking is often portrayed as the mark of a blase and sophisticated individual, constituting a strong attraction for adolescents. The commercials constantly extol the joys of smoking and the absence of any harmful effects. These claims are often bolstered by the reports of surveys of physicians who have found no adverse effect of smoking. Even "Nose and Throat specialists" are included as certifying to the absence of any irritating qualities in a particular brand of tobacco. This type of advertising must be disturbing to laryngologists who so frequently see throat conditions which they attribtute to smoking and who attempt to convince the patient to stop the habit.

This presentation aims to review the diseases of the larynx that are considered to be the result of smoking and to evaluate the evidence that serves to indict tobacco. The laryngeal conditions most often blamed on smoking are: *1.* chronic nonspecific laryngitis; *2.* polypoid degeneration of the vocal cords; *3.* leukoplakia or keratosis, and *4.* carcinoma.

CHRONIC NONSPECIFIC LARYNGITIS.

This is an exceedingly common disease. Numerous factors may contribute to its development, such as dusty air, abuse and faulty use of the voice, infection of the sinuses or the chest, with chronic cough, mouth breathing, alcohol, as well as tobacco smoke. Actually, in routine office practice the condition is so commonly seen in heavy smokers that this seems the single most important and common etiologic agent.

The gross appearance of the larynx is characteristic. There is hyperemia of the mucosa so that it is darker red and furry in appearance. Thickening of the mucosa is evidenced by a rounding of the arytenoids and blunting of the sharpness of the landmarks. The smaller capillaries may be dilated. The true cords do not appear as white, flat bands, but are rounded, thickened, and hyperemic, with visibly dilated vessels. The edges may be irregular with sticky adherent secretion.

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[*] Read at the meeting of the Middle Section of the American Laryngological, Rhinological, and Otological Society, St. Louis, Mo., January 18 1954.

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The patient may not be aware of an alteration of the voice, or of hoarseness, or complain of any symptoms referable to the throat. Chronic laryngitis is a common incidental finding in the routine examination of heavy smokers. When there are no subjective complaints, the chief significance of its existence would seem to be in the possibility of chronic laryngitis acting as a precursor of more serious disease. Speaking with chronically inflamed vocal cords may lead to voice strain that results in the formation of polyps or of polypoid degeneration. The prolonged irritation of the mucosa may cause keratosis or even malignancy.

The evidence that smoking is an important etiologic factor in chronic laryngitis is based on: *1.* the conclusions of most clinicians with the widest experience; *2.* the well established fact that the condition is usually reversible; when the irritation of smoking is removed, the larynx often returns to normal.

POLYPOID DEGENERATION OF THE CORDS.

In 1950 Myerson[1] described this lesion of the vocal cords as a clinical pathological entity directly due to smoking. His conclusions were based on the study of 143 patients with edema or diffuse polypoid changes of the anterior two-thirds of the true vocal cords. His illustrations reveal five different stages, from simple edema to bilateral rounded polypoid masses with a sessile base but freely movable because of their thin attachment. He described the histology as an edematous fibroma. The epithelium may vary somewhat, but usually is the normal stratified type of the free edge of the cord. The bulk of the tumor consists of fibrous tissue in an edematous stroma. Myerson postulates that the local irritation of smoking first results in an outpouring of serum into Reinke's space. As a result of this chronic edema there is increased fibrosis, and finally a polypoid tumor develops.

His evidence for concluding that this polypoid formation was directly due to smoking was: *1.* all of his 143 patients were heavy smokers; *2.* his observation that in the early stages when only edema was present the swelling would disappear within 24 hours when smoking was stopped; *3.* his histologic studies of the various stages in the development of these growths.

Jackson[2] termed this condition polypoid corditis. He described it as a frequent finding in chronic laryngitis. He thought these tumors were of inflammatory origin, and described them, along with pedunculated polyps, as edematous fibromata. Holinger[3] described this condition with the term polypoid degeneration of the cord, under the classification on non-neoplastic tumors. He reported on 112 patients with this disease, and found the histology similar to preduncuIated polyps.

Putney[4] and Clerf called the disease a chronic hypertrophic laryngitis and concluded that it was due to vocal abuse or infection. They advised stripping of the cords. They also found the chief histologic alterations as edema and fibrosis.

Since Myerson's report, twelve patients with polypoid degeneration of the cords have been studied. Eleven were smokers; one had never smoked but had used snuff for many years. The gross appearance was as described by Myerson (see Figs. 1, A, B, C, D, 2C). In five of the patients the growths were removed, and examined microscopically. The findings were essentially as described above. On comparison with sections made of pedunculated polyps, a similar histopathology was manifest (see Fig. 2, A, B, D, C).

Removal of the growths was not advised in these patients until the patients had given up smoking. In one patient this resulted in a complete disappearance of large bilateral polypoid masses. The improvement did not occur rapidly, within 24 hours as described by Myerson, but took place gradually over several months.

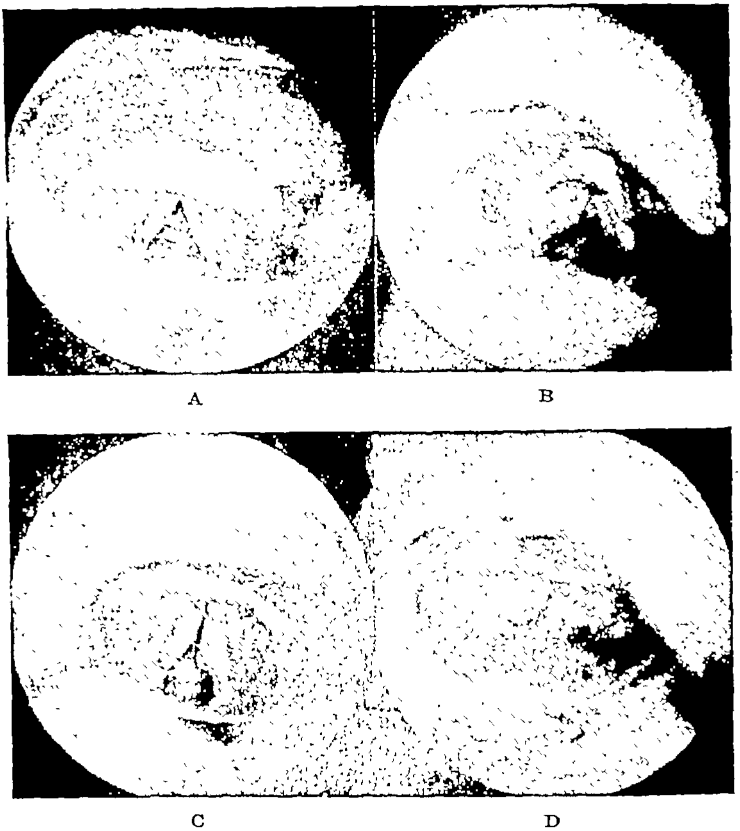

Fig. 1. —A. Photograph of the larynx of a male excessive smoker revealing diffuse edema of the anterior half of both cords.

B. Polypoid degeneration of the right cord of a female non-smoker who had used snuff for many years.

C. Male heavy smoker and constant talker. There is polypoid degeneration of both cords, with superficial leukoplakia of the right.

D. Marked polypoid degeneration of the anterior half of both cords in a male excessive smoker.

REPORT OF A CASE.

A 72-year-old male executive was seen because of hoarseness following a cold of 10 days' duration. He had frequent episodes of hoarseness treated by local applications, had been a heavy cigarette smoker for many years, and talked a great deal over the long distance telephone as well as in conferences. Mirror laryngoscopy revealed marked polypoid degeneration of the anterior two-thirds of both true cords. The masses were pale and edematous in appearance. They were so large they occluded the anterior portion of the glottis, but were very mobile, moving up and down on respiration. There was diffuse hyperemia of the remainder of the larynx. The condition was described to the patient and he was urged to stop smoking, which he did. Within two weeks there was considerable decrease in the size of the ploypoid enlargements. After three months' observation, the cords were normal in appearance and the patient's only complaint was his weight gain since stopping smoking.

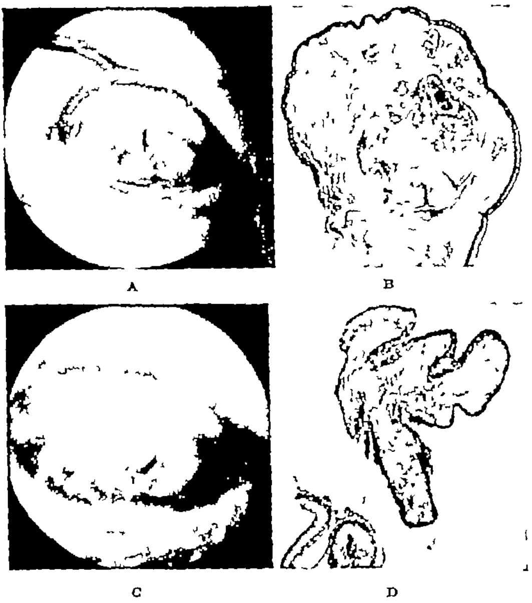

Fig 2—A Typical pedunculated polyp of the right cord.

B. Photomicrography of the polyp removed from the patient in 2—A. There are only minor alterations of the mucosa. The bulk of the tumor consists of an edematous stroma with fibrosis There is a large round area of hyaline.

C Male excessive smoker, drug addict and constant talker. There is marked polypoid degeneration of the right cord.

D Photomicrograph of the tissue removed from the patient, 2—C. There is thickening of the epithelium The stroma is edematous with dense fibrosis. (Compare with 2 B)

Pedunculated polyps (see Fig. 2A) of the vocal cords are known to occur in non-smokers. Their formation is usually attributed to vocal abuse. Diffuse polypoid degeneration of the cords has a similar histopathology, and it would seem inconsistent to consider them due to smoking and the discrete polyps to vocal strain. The similar histopathology suggests a similar etiology. This common denominator may well be vocal strain, whether from vocal abuse, or from speaking with the constantly inflamed cords of chronic laryngitis due

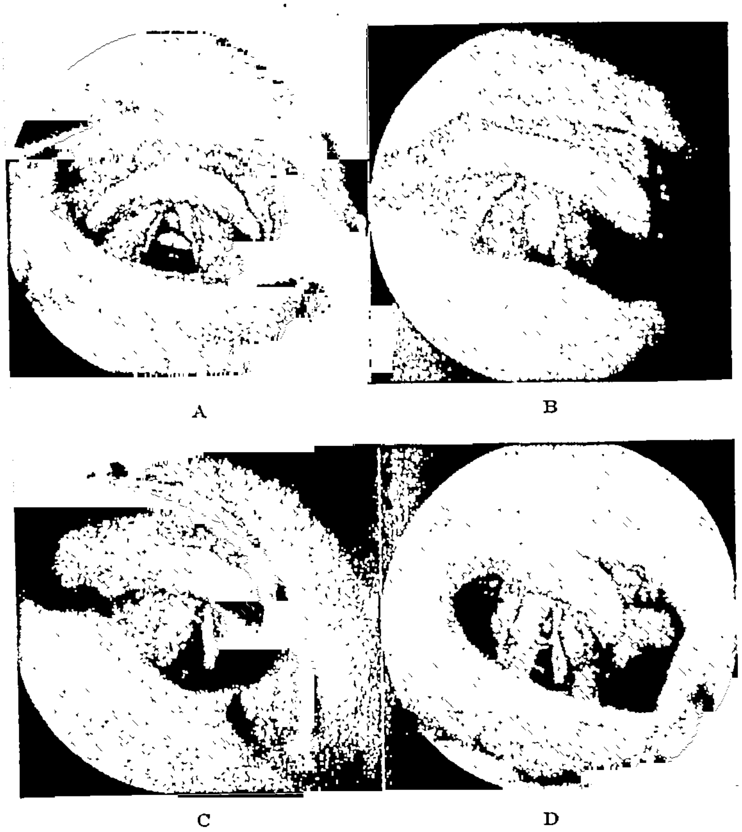

Fig. 3.—A. Male heavy smoker with keratosis of the anterior half of the left cord.

B. Marked keratosis of the left cord of a male excessive smoker.

C. Keratosis and infiltration of the right cord of a male excessive smoker. Biopsy: Carcinoma arising in leukoplakia.

D. Keratosis and infiltration of the right cord of a male heavy smoker. The right cord had been stripped of leukoplakia six years previously. Biopsy: Carcinoma.

smoking. Friedberg[5] and Segall studied Reinke's space and its relation to cordal polyps. They explain the usual site of discrete polyps at the junction of the anterior and middle thirds as due to the existence of a "vibration node" at this site. If the outpouching does not occur at this point, a sessile type of polyp results. This concept does not detract from the importance of smoking in the etiology of polypoid degeneration, but rather explains that it is not necessarily a specific effect.

KERATOSIS, HYPERKERATOSIS AND LEUKOPLAKIA.

These terms are used synonymously by many authors and as distinct entities by others. Jackson[8] and Jackson describe leukoplakia and keratosis as separate diseases. New[16] and Erich consider them as distinct though similar conditions. Clerf,[7] Putney[4] and Holinger[5] use the two terms as indicating the same disease. As used in this discussion, the term keratosis denotes the chalky white localized elevations of the mucosa, which histologically reveal benign epithelial hyperplasia, without invasion of the submucosa (see Fig. 3, A, B). There is usually an associated chronic laryngitis, causing the whitish areas to contrast all the more with the hyperemic mucosa. A chronic irritation is usually advanced as the reason for the development of keratosis, with smoking listed as the commonest offender. Clerf[7] states that he is convinced that smoking is a predisposing factor, but admits it has not been proven. He is impressed with a deficiency of Vitamin A as an additional factor in some patients. Putney[4] and O'Keefe state that smoking has long been considered a salient factor. In their series of 125 patients with keratosis of the larynx only 11.2 per cent were in non-smokers, compared with 34 per cent of non-smokers in the general population. LeJeune[9] believes that keratosis is being seen more commonly these days. All of his patients with the disease used cigarettes excessively. In a recent study by Friedberg[10] and Wallner, all patients with benign keratosis were heavy smokers.

REPORT OF A CASE.

A 40-year-old male was referred for examination of the sinuses as a possible cause of a chronic cough. He complained of postnasal discharge and hoarseness for three weeks. He admitted smoking two packs or more of cigarettes daily. On examination the nasal airways were adequate, the mucosa was quite hyperemic. The sinuses were moderately clear on transillumination and X-rays revealed them to be normal. The tonsils had been cleanly removed. The larynx revealed diffuse leukoplakia of the anterior third of each true cord. There was intense hyperemia of the rest of the larynx. The patient agreed to stop smoking, and no other treatment was given. In three weeks he reported his voice greatly improved and less postnasal discharge. The leukoplakia was less marked. Two months later the leukoplakia had disappeared and only a slight hyperemia remained. Six months after the first examination he reported that his cough and postnasal discharge had completely disappeared and that his voice was normal. His larynx appeared normal.

We may summarize the evidence that smoking is an important factor in the etiology of keratosis: *1.* this is the conclusion of all writers, based on their clinical experience, *2.* keratosis is often reversible in that when smoking is stopped, the condition may disappear, or is less likely to recur after removal.

CARCINOMA.

The etiology of cancer remains an enigma, but chronic irritation is frequently listed as an important factor. It is a common observation that malignant disease of the larynx is often preceded by long periods of hoarseness. Jackson[2] states "Precancerous conditions are present in probably 75 to 80 per cent of the cases . . . as a clinical fact we have rarely found cancer developed in a normal larynx." Szanto, a pathologist, writing in Lederers'[11] text says, "Cancer rarely develops in a normal larynx . . . in at least half of the cases of laryngeal cancer, precancerous lesions antedate the malignant growth" (see Fig. 3 C). "Many writers have emphasized the importance of precancerous lesions in the development of laryngeal carcinoma. Precancerous and malignant growths may have similar gross appearance, they may be difficult to differentiate histologically, and a lesion that was regarded as benign may later become malignant." Clerf, Putney and O'Keefe, and New and Erich describe observing patients with benign keratosis for long intervals and finally finding evidence of carcinoma (see Fig. 3 D). Friedberg[10] and Wallner found of 39 patients with cordal cancer, 19 or 61 per cent had leukoplakia of the vocal cords.

It thus becomes obvious that if we accept smoking as an etiologic factor in keratosis, we must also regard it as a factor in the development of cancer. Even in those patients with laryngeal cancer without known precancerous lesions, there is evidence that heavy smoking plays a role. In the series mentioned, studied by Friedberg[10] and Wallner, of 116 patients with laryngeal cancer, there were 97 known smokers, 3 non-smokers, and in 16 the information was not available. This incidence of smokers is appreciably higher than the 73.7 per cent figure quoted by Wynder[12] and Graham as representing the smokers in a general (non-cancer) population.

There have been a number of other careful statistical surveys on the relation of smoking to cancer of the respiratory tract.[13,14] These reveal a significantly higher percentage of smokers among those with cancer than among the control groups.

The evidence that smoking is a factor in the development of laryngeal cancer is circumstantial. It is based on the judgment of most clinicians with the widest experience, who have observed the high percentage of smokers among patients with laryngeal malignancy.

TREATMENT.

One of the fundamentals in the management of chronic laryngeal disease is for the patient to stop smoking. This seems a simple enough remedy, but in practice it is most difficult to accomplish. Most patients will offer to "cut down" on their consumption, or to stop for awhile. The prospect of permanently abolishing the habit seems to appall them. It is a question whether it is better to insist on complete abstinence, with less chance of success, or to compromise and permit a greatly curtailed amount of smoking, as described by Jackson.[2] Obtaining the patient's cooperation is certainly a difficult problem, taxing the physicians' power of persuasion, salesmanship and patience. It requires a great deal more time and effort to convince the patient of the necessity of not smoking than to administer some local treatment. The physician's efforts may be rewarded by observing the improvement in the larynx, and in the gratitude expressed by the patient for his general improvement and sense of well being.

COMMENT.

This review indicates that there is no one disease of the larynx that has been proven as specifically and entirely due to smoking. Rather, excessive smoking may be considered the chief, though not the only factor in three diseases, and probably important in the fourth. Based on frequency of occurrence, chronic laryngitis could be considered as the typical "smoker's larynx," though keratosis is more likely to be thought pathognomonic of excessive smoking.

One may ask, what would constitute definite proof of the etiology of a disease? Animal experimentation gives only corroborative evidence, though the results of the work of Wynder,[15] Graham and Croniger with tobacco tar producing skin cancers in mice seem as applicable to cancer of the larynx as of the lung. The fact that a disease may have more than one cause is not proof against any given agent. The best evidence we have at present is clinical, the fact that we can often reverse the disease process when the irritant is withdrawn. This is true for chronic non-specific laryngitis, and to a lesser degree for leukoplakia and polypoid degeneration of the cords.

Accurate statistics are not available, but chronic non-specific laryngitis is certainly very common in individuals who have smoked excessively for years. The patient may not be aware of any abnormalities in his voice and have no throat complaints. Alterations in the voice may not be as noticeable in men as in women, where a huskiness or lowering of pitch is more obvious and objectionable.

Considering the number of persons that smoke, the more serious forms of laryngeal disease are not common. Even if we accept the higher percentage of smokers among patients with laryngeal cancer as significant, it does not tell the other aspect of the question, how dangerous is smoking? Mortality statistics of the U. S. Census Bureau[16] lists 0.76 per cent of deaths of men past 40 due to cancer of the respiratory tract. This would indicate that even if tobacco smoke is a carcinogenic agent it is a weak one. It is doubtful if we are justified in using fear of cancer alone in attempting to curtail a habit that seems to bring so much satisfaction to so many millions. Scientific accuracy would require us to stress instead the less serious but common irritative conditions associated with smoking.

In any discussion of the harmful effects of smoking it must be remembered that the larynx is only one organ that may be adversely affected. To obtain the complete picture, we should consider the effect on the nose; in decreased olfaction, blocking and nasal and post nasal discharge. There may be a chronic pharyngitis with dryness and sore throat. The possible relation to cancer of the lung is receiving wide publicity. The chronic "smoker's cough" is well known. The effects of smoking on the digestive system, heart and circulatory systems are more controversial; however, when the overall effects of smoking are studied, deleterious results are not uncommon. Once acquired, the habit usually remains with an individual. Many of the adverse effects are not noticed until after years of smoking. Graham and Wynder[12] found the carcinogenic effect in the lung occurred after 30 to 35 years, or one-half the life span. This was substantiated by their animal experiments.[15] Short term surveys, purporting to discover no ill effects after a few months' observation, are apt to be misleading.

The results of polls of physicians as expressed by the tobacco companies may not be as significant as the conclusions of the smaller number of investigators who have studied the problem more intensively.

SUMMARY AND CONCLUSIONS.

The irritation of tobacco smoke is a frequent cause of chronic laryngeal disease. Smoking is an important factor in chronic non-specific laryngitis, in polypoid degeneration of the vocal cords and in keratosis. Statistical evidence also indicates it is a factor in the etiology of laryngeal cancer.

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AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1 and continue for a period of ten months. Registrations must be completed before August 15. Detailed information and application forms may be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minnesota.

The Alexander Graham Bell Association for the Deaf (formerly The Volta Speech Association for the Deaf) will hold its 1954 Summer meeting in St. Louis, June 14-18 inclusive. The theme of this meeting is "Let's Face the Issues," and some of the questions to be discussed are: "Where should the deaf child receive his education?" "What are the possibilities and limitations of auditory training?" "What are the principal issues in teaching speech?" and "What contributions can be made to the understanding of the deaf child by persons in peripheral fields?"

All sessions will be held at the Chase Hotel, but there will be Open House at Central Institute for the Deaf and St. Joseph Institute for the Deaf.

For further information, address: Robert Goldstein, 818 S. Kingshighway, St. Louis 10, Missouri.

NON-SURGICAL MANAGEMENT OF MENIERE'S DISEASE.[*]

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The voluminous literature on the symptom complex of Meniere's disease has not clarified its pathogenesis nor has it positively demonstrated more of the pathology in the inner ear than the dilatation of the endolymphatic duct. Obviously, any plan of treatment of the disease has been and still is empirical. That such empirical management leaves something to be desired is only too apparent in reviewing even the literature of the last few years, and then in applying the suggestions for treatment in one's own practice.

To avoid confusion in terminology and symptomatology with other authors, the following clinical picture has been our basis for a diagnosis of Meniere's disease or "labyrinthine hydrops." These terms are used synonymously for the symptom complex of a low tone perceptive deafness that usually fluctuates widely, at least in the early stages; a roaring type of tinnitus; complaint of a sense of pressure or fullness in the ear, and paroxysmal attacks of vertigo. Hyperacusis not infrequently is present and often the symptom of diplacusis can be demonstrated. This is a relatively easy diagnosis to make, and it is rarely a diagnostic necessity to have the results of such special functional tests of the labyrinth as recruitment and difference limen tests, discrimination scores with the P. B. List, or even caloric tests of equilibrium.

Not all of the preceding symptoms need to be present to justify a diagnosis of Meniere's disease. More often than not the entire symptom complex is present, but less frequently one sees either the picture of cochlear or vestibular symptoms alone.

PATHOGENESIS.

To me the most appealing descriptions of the possible etiology and pathogenesis of Menier's disease, based upon keen

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clinical observations and the application of the knowledge of basic physiological studies of the autonomic nervous system, are those of Hilger[1,2] and Williams.[3,4] Hilger[1,2] represents the various clinical syndromes of inner ear pathology as resulting from autonomic dysfunction in the labyrinthine neurosvascular sphere. This dysfunction produces angiospastic change in the labyrinthine arterial supply and, thus, labyrinthine ischemia with a resultant transudate from ischemic damage to the capillaries. In this way variable cochlear and vestibular involvement can occur, the differences being due to the degree of ischemia and the nature of labyrinthine elements involved.

Williams[3,4] states that a localized autonomic dysfunction producing a stereotyped vaso-spastic vaso-atonic condition in the peripheral vascular bed is the basis for the development of pathologic changes in Meniere's disease. He feels that autonomic dysfunction and allergy are synonyms describing identical phenomena. The arteriolar spasm with capillary and venule dilatation causes anoxia of the capillary loop, injury to the endothelium and increased permeability of the capillary loop. This type of involvement of the stria vascularis results in an increased volume of endolymph with an increased protein content and higher osmotic pressure producing the typical cochlear symptoms. Williams states that the vertiginous crises are produced by vasospasm of the branches of the internal auditory artery supplying the maculae and cristae which causes local anoxia, or by a sudden release of vasospasm producing irritation from a sudden resumption of blood flow.

THERAPY.

Authors have varied considerably in selecting the chief instigating factors of autonomic dysfunction to attack in their rationale for therapy of Meniere's disease. The narrow selection of precipitating factors is exemplified by the approach of such writers as Wright[5] in his focal infection theory; Selfridge[6] and others with their avitaminosis theory, or Gundrum[7] in a recent report suggesting an allergic etiology in 34 of 57 non-traumatic cases of Meniere's symptom-complex. Mygind and Dederding,[8] and Furstenberg and associates[9] were others who gave principal emphasis to one factor, considering respectively the water balance or the sodium retention as the single most important factor to be controlled in the treatment of Meniere's disease.

My own feeling is that numerous factors must be considered as possible precipitators of the crisis of localized autonomic dysfunction in the inner ear. Any one of these, or a combination of several, can be the trigger in a given case. The relative importance of the different factors is difficult to evaluate, but the broader viewpoint of etiology is more in keeping with that expressed by Williams or Hilger.

In attempting to evaluate an individual patient with Meniere's disease we try, through a careful history, to elicit evidence of the following: any emotional or stress influence; tobacco, alcohol, salt, fluid or specific dietary over-indulgence; medications taken over a long period, including hormones and vitamins; coincidence between severe upper respiratory infections or influenza with attacks; apparent variation with menses, season of the year, climate, trips or physical environmental contacts; clinical manifestations of specific antigen-antibody allergy, such as in seasonal or perennial allergic rhinitis, bronchial asthma, eczema or allergic gastro-intestinal disorders, other vascular phenomena such as classical and atypical histaminic head and neck pains.

Any one of the above mentioned factors has been the only important one in one or more cases in our practice, and a combination of several such factors has been even more common; however, unfortunately in our experience, quite frequently none of the factors listed can be proved, or even strongly suspected, in certain individual cases. The most rewarding single etiologic lead which we have been able to establish has been a specific antigen-antibody reaction in approximately 5 to 10 per cent of the cases of Meniere's disease seen in our office in a given year. The most completely unrewarding search has been for foci of infection or vitamin deficiencies.

In outlining our usual medical management of the patient with Meniere's disease, I am concentrating on the office treatment rather than the occasional acute vertiginous or cochlear crisis necessitating hospitalization. As stated earlier, an attempt is made to establish any fairly definite precipitating factors by a thorough history, examination and finally by a therapeutic test. The therapeutic test, in this instance, is the relief of symptoms by the removal of the offending factor or by appropriate treatment of the factor, and the recurrence of symptoms upon re-exposure to the factor, or by stopping treatment. Applying this same approach to establishing specific allergic etiology is the reason for our confidence in the control of 5 to 10 per cent of our cases of labyrinthine hydrops by specific allergic management. The most commonly missed specific allergic factor is apt to be one of the staple foods, unless it is unmasked by specific feeding tests, according to Rinkel's[10] technique.

If we fail to establish or to control the precipitating factors, or if such an attempt is apt to be a long drawn-out procedure, in which eventual success is uncertain, then what I term nonspecific management is begun.

Our modification of the "salt-free" Furstenberg diet is shown in Fig. 1. I find that by liberalizing the diet to allow salted butter and the ordinarily prepared bread, with the use of the proprietary salt substitutes, most patients will adhere to the program. This amounts to approximately a 1.0 gm. sodium diet. To this is added potassium chloride, two teaspoons of a 25 per cent aqueous solution three times daily during meals for three consecutive days, alternated with two days of omission. The employment of "salt-free" diets and ammonium or potassium chloride has become almost a ritual in the management of labyrinthine hydrops, and certainly this ritual needs drastic re-evaluation in the face of the recent report by Perlman and his associates[11] of their complete inability to produce a consistent effect upon cochlear or vestibular function in fifteen patients with active Meniere's disease, while on hospital programs which produced measured increases or decreases of serum sodium levels.

Fig 1

FURSTENBERG DIET.

GROUP A—The following foods may be taken daily
1. Eggs, meat, fish and fowl as desired, without salt.
2. Bread as desired, without salt.
3. Cereal, one of the following Farina, oatmeal, rice, puffed rice, puffed wheat.
4. Potato and one or more servings of any of the following: a) macaroni, b) spaghetti, c) rice, d) corn, e) cranberries, f) prunes, g) plums.
5. Milk as desired
6. Vegetables and fruits daily of any fruit and of any vegetables not included in groups "B" and "C" as desired
7. Butter, cream, honey, jellies, jams, sugar and candy permitted as desired.
8. Tea and coffee as desired.

GROUP B—Foods to be avoided
All salt meats and salted fish, or bread, crackers and butter prepared with salt. Carrots, clams, condensed milk, raisins, caviar, cowpeas, olives, spinach, cheese, endive, oysters

GROUP C—Foods to be taken no more than twice weekly
Lima beans, beets, buttermilk, cantaloupe, cauliflower, celery, chard, dried coconut, dried currants, dates, figs, horseradish, kohlrabi, limes, muskmelons, peanuts, peaches, mustard, pumpkin, radishes, rutabagas, strawberries, turnips, turnip tops, watercress

NOTE All Foods to Be Prepared and Served Without Salt.
Water intake unrestricted although excessive quantities of liquid should not be taken.

MEDICATION. Potassium chloride, 2 tsp., 25% solution, t.i.d., alternate 3 days of intake with 2 days of omission

SALT SUBSTITUTE Diasal

Most authors are agreed upon the need of using a vaso-dilating agent to combat the basic functional lesion of vasospasm. The disagreement that exists in this type of therapy is chiefly in the specfic vasodilator used, and somewhat in the route, amount and frequency of administration. Of the smooth muscle dilating agents our preference is histamine. I feel that the effect of histamine is probably secured by its reaction upon the spastic-atonic state of the peripheral vascular bed, rather than by a so-called "desensitization."

We make a consistent attempt to individualize the histamine therapy by varying the dosage according to the patient's reaction or lack of reaction. This is the "small optimum dosage of Hansel" in contrast to the usually reported much stronger increasing dosage technique of subcutaneous administration, or the constant intravenous dosage. The intravenous dosage usually amounts to 1 cc. of 1:1000 dilution of histamine given daily at a slow drip rate. The build-up therapy consists of twice daily subcutaneous injections of 1:10,000 dilution, up to the 1:1000 dilution. It is unfortunate that the criticisms of the results of histamine therapy are based upon the latter two types of administration, and are often made by the same authors who advocate a small optimum dosage method for giving either subcutaneous monoethanolamine nicotinate (nicamin) or oral nicotinic acid.

The optimum dosage technique as we employ it begins with 0.1 cc. of a 1:100,000,000 dilution of histamine diphosphate solution given subcutaneously. If no noted effect follows within 24 hours then the dosage is increased one dilution to 0.1 cc. of a 1:10,000,000 dilution given within one to three days after the first injection. The subcutaneous histamine dosage is increased in this manner until the optimal dosage, or that amount which will just relieve the symptoms is reached. Once the dilution has been found which will give a day or so of improvement, the dosage is increased by making the amount of that dilution greater; for example, from 0.1 cc. to 0.25 cc. to 0.50 cc., etc. The ultimate best results are found to be on a subcutaneous dosage which maintains the patient symptom-free for intervals of three to seven days, supplemented by sublingual administration of histamine one or two dilutions stronger than the subcutaneous dosage, using two drops twice daily. If the initial dosage of 0.1 cc. of the 1:100,000,000 dilution precipitates a Meniere's attack, then the dosage is lowered to 1:10,000,000,000 and further adjustments are made to obtain an optimum dose.

When the patient under non-specific treatment has been free of vertigo, and the hearing has been stabilized at the highest possible level without the symptoms of distortion and pressure for a period of six to eight weeks, the histamine and potassium chloride are discontinued, and the salt-free diet is liberalized. Any recurrence of symptoms is followed by the resumption of the complete program.

I feel that one can best judge that a given histamine injection has been effective by an almost immediate (within several minutes to a few hours) improvement in cochlear symptoms. The repeated improvement from an injection can be very convincing to the patient and to the physician. Any improvement occurring only after weeks and months of the same level of therapy, whether it be histamine or any other drug, is more likely due to the spontaneous remission from "nature's" tendency to re-establish autonomic balance than from a therapeutic result.

EVALUATION OF RESULTS OF TREATMENT.

It is difficult to evaluate the over-all results of treatment of Meniere's disease. No standard criteria have been established for such an evaluation. Too often the spontaneous remissions of the disease gain unjustified credit for the coincidental therapy. Furthermore, we must depend so frequently upon the subjective evaluation of the variation in symptoms by a wishful and anxious patient. The various hearing tests with tuning forks, audiometry and the more specialized tests for recruitment and discrimination, provide the best objective evidence for evaluation of cochlear changes; however, unless one has such records of a patient's hearing prior to the development of labyrinthine hydrops, there is uncertainty as to whether treatment has completely reversed the reversible cochlear pathology.

The above mentioned difficulties in evaluation became particularly apparent when I attempted to appraise the treatment received by 55 new cases of Meniere's disease in the common otologic practice of Dr. George E. Shambaugh, Jr., and myself from July 1, 1952, to July 1, 1953. For that year 362 new cases of perceptive deafness were reviewed, selecting 55 cases of unquestionable Meniere's disease. Of these 42 had reasonably definitive results to evaluate, and 13 were inconclusive, due either to an inadequate trial of therapy or to inadequate records. The changes effected in the principal cochlear and vestibular systoms were classfiied as completely relieved, moderately improved, slightly improved, and not relieved at all. Complete relief implied disappearance of any vertigo, and the previously fluctuating hearing loss stabilized at a normal level and absence of tinnitus and aural pressure. Moderate improvement meant much less frequent and severe attacks of vertigo, and the hearing stabilized at a significantly improved level, while tinnitus and sense of pressure have become minimal and possibly intermittent. Slight or no relief in the various symptoms hardly needs explanation.

Thus the varied relief of the individual symptoms is as follows:

VERTIGO: Of the 42 cases, 34 had vertigo; 18 showed complete relief, and five were moderately improved, totalling 23 or 68 per cent.

IMPAIRED HEARING: A significant loss in the speech frequencies of 15 decibels or over was present in 35 cases, of which only six showed complete relief and nine were moderately improved, totalling 15 or 43 per cent.

TINNITUS: Of 34 patients with tinnitus, nine had complete relief and nine were moderately relieved, a total of 18 or 53 per cent.

FULLNESS OF PRESSURE: This was the least common symptom, occurring in 28 patients of the 42. Of these 12 had complete relief and six had moderate relief, a total of 18 or 64 per cent (see Table I).

An interesting comparison between the above percentages of relief can be drawn with a report by Shambaugh[12] on histamine treatment of a labyrinthine hydrops-like syndrome in fenestrated ears, months or years after surgery. The respective percentages of relief in a much longer series of 370 patients for the symptoms of vertigo, drop in hearing and tinnitus were 65, 35 and 60 per cent.

Of the 42 cases, 25 had the complete triad of symptoms of Meniere's disease.

Six cases were classified as allergic in etiology, and in each one specific allergic management resulted in moderate or complete relief of all symptoms.

Only one case in this group of 55 cases underwent a labyrinthotomy because of incapacitating vertigo. After six months of complete relief, this patient has developed tinnitus, pressure and impaired hearing in the opposite ear, which is responding only partially to dilute histamine.

TABLE I.

RESULTS OF NON-SURGICAL MANAGEMENT OF MENIERE'S DISEASE IN 42 PATIENTS—BY SYMPTOMS.

Vertigo	34 Cases	68 Per Cent Relieved or Improved.
Tinnitus	34 Cases	53 Per Cent Relieved or Improved.
Hearing Loss	35 Cases	43 Per Cent Relieved or Improved.
Aural Pressure	28 Cases	64 Per Cent Relieved or Improved.

CASE REPORTS.

The audiograms in the case reports are shown without bone conduction recordings; however, these tests along with the tuning fork results of positive Rinne's and shortened Schwabachs in the involved ears all were consistent with the diagnosis of a perceptive deafness. Although the discrimination scores are recorded only in the first case report, all these patients gave descriptions of their hearing being "out of tune," "distorted" or "fuzzy." Since most otologists still do not do routine discrimination tests in their office practices, reliance upon such subjective comments as an indication of impaired discrimination is justifiable.

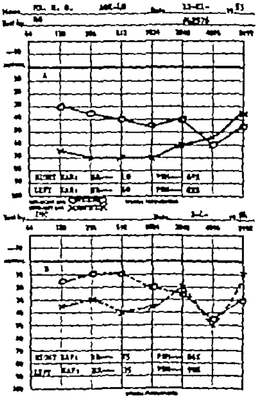

Fig. 2.

Case 1.—H. O., a man aged 40, was referred to me by his psychiatrist who desired an opinion as to whether a bilateral hearing loss of three months' duration was psychogenic or organic. A careful history defined the hearing loss as gradual in onset and progression rather than fluctuating in character. No vertigo or tinnitus were noted; however, the patient, an excellent pianist, volunteered that he noted considerable difficulty in pitch perception, as though he were "out of tune."

Examination revealed normal ear, nose and throat findings. The tuning fork findings confirmed the air and bone audiometry (see A in Fig. 2) which revealed a bilateral relatively flat perceptive loss, greater in the left ear. Diplacusis was demonstrated with the 256, 512, 1024 and 2048 tuning forks heard as higher pitched in the left ear. The discrimination scores, using the P B Max test, were quite low in both ears (see A in Fig. 2).

Except for a possible emotional factor, no obvious etiology was evident. The salt-free diet, potassium chloride and histamine management were started. The patient reported that "the right ear opened up" within an hour after receiving 0.1 cc. 1:100,000,000 histamine subcutaneously, and on the following day the "left ear opened up." Sublingual histamine, two drops twice daily, and a twice weekly histamine injection maintained the improvement in hearing, and the ears were no longer out of tune. This improvement is reflected in both the pure tone audiogram and the discrimination scores (see B in Fig. 2).

Comment: This case shows that treatment for a very possible etiologic emotional factor in cochlear hydrops by the psychiatrist did not obviate a trial of dilute histamine therapy, which we may conclude gave a positive and immediate response. Naturally, I realize that the case is too recent to evaluate as to whether it will be a maintained successful result; however, such a quick response to histamine, in my experience, augurs well for the eventual control of symptoms.

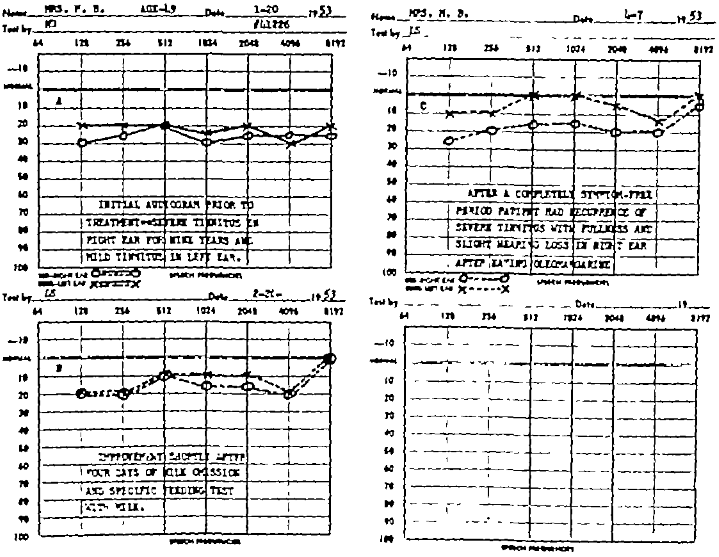

Fig. 3.

Case 2.—M. B., a married woman aged 49, was first seen on January 20, 1953, complaining bitterly of a constant tinnitus "like an African jungle or a jet plane" in the right ear for the previous nine years. This severe tinnitus often interfered with sleep and completely overshadowed the other symptoms of a fullness in the right ear, a recent mild tinnitus in the left ear, and a "possible" hearing loss (see A in Fig. 3). No vertigo has been experienced. Previous treatment had been surgery "for scar tissue in the back of the nose and throat," inflations and sedation.

The examination revealed normal drum membranes and middle ears. Audiogram (see A in Fig. 3) revealed a mild bilateral perceptive loss with positive 256 Rinne's. No diplacusis was determined. Additional history was obtained of a constant, non-seasonal post-nasal discharge, but sinus X-rays were normal. There were numerous food and drug sensitivities elicited. Pork caused severe headache. Oranges, tomatoes and chocolate caused hives. Penicillin caused hives. Was once given a histamine (dosage ?) injection which caused fainting, hives and a watery nasal and eye drainage. As a child on the farm, the patient drank large amounts of milk, and now has indigestion from milk, so uses small amounts of milk, cream and butter.

In spite of a negative intradermal skin test to milk, the patient began a complete milk omission diet on February 18, 1953, and did a specific milk feeding test on the fifth day. Within ten minutes after the feeding of milk the patient developed sneezing, watery nasal drainage, coughing, nausea, cramps, a full feeling in the head and an increased "pounding" tinnitus. The audiogram three days later showed improvement in hearing (see B in Fig 3).

Complete milk omission has given complete relief from the tinnitus, and normal hearing, except for one occasion when patient ate oleomargarine which contains milk, and all symptoms recurred (see C in Fig. 3).

Comment: Two doses of dilute histamine were tried before the milk feeding test with mild reduction in severity of tinnitus lasting about two days after the injection; however, milk omission resulted in such complete relief that no further histamine was used. At the present time the patient still finds that she cannot tolerate even small amounts of milk.

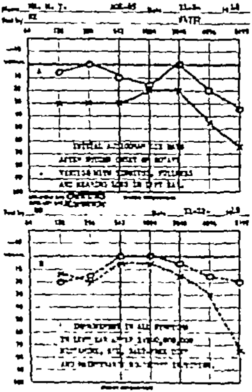

Fig. 4.

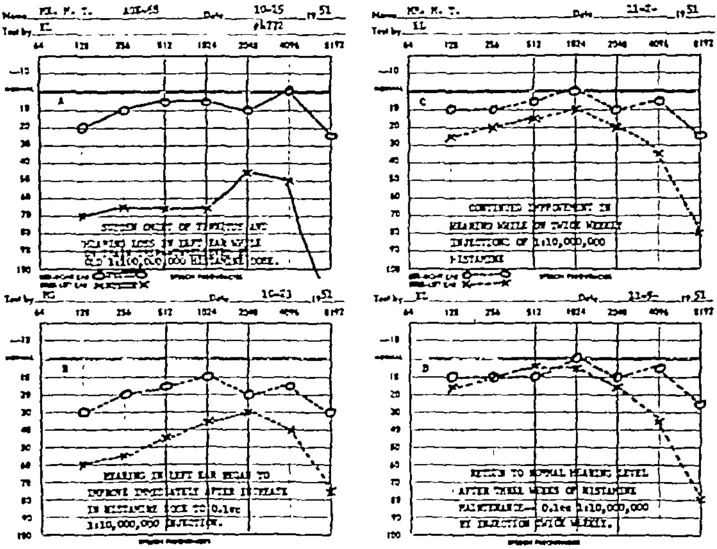

Fig. 5.

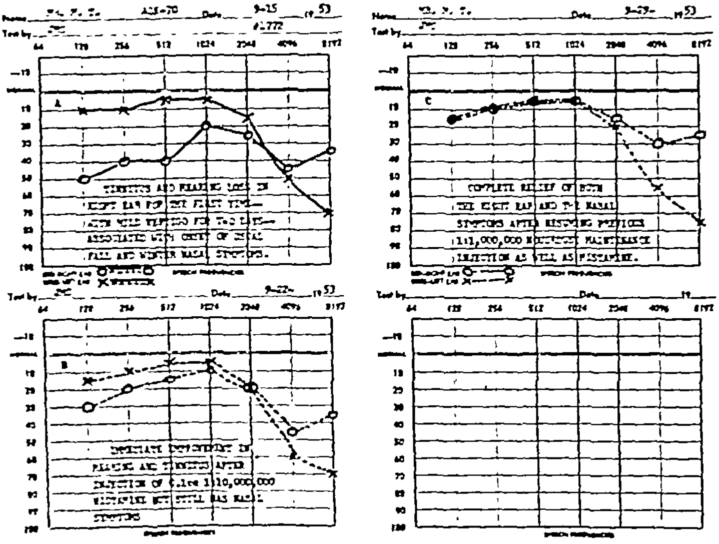

Fig. 6.

Case 3.—M. T., a man aged 70, has been a patient of the Shambaughs, Sr., and Jr., since 1915 because of a chronic yearly winter time recurrence of nasal blocking, nasal and post-nasal drainage, frontal and maxillary pain. On many occasions upper respiratory infections resulted in maxillary sinusitis necessitating irrigation. Following the realization of the importance of a sensitivity to house dust and the giving of house dust injections, the sinuses have not needed irrigation.

On November 2, 1948, the patient developed his first attack of tinnitus and impaired hearing in the left ear, followed the next day by a two-hour attack of rotary vertigo with nausea. The audiogram (see A in Fig. 4) and the tuning forks showed a perceptive deafness in the left ear particularly. In addition to his usual house dust injection, 0.1 cc. of 1:100,000,000 histamine was given. After the attack of vertigo on the following day, the tinnitus and hearing loss receded with a return to near normal hearing (see B in Fig. 4).

The patient had no particular difficulty, and in subsequent winters got along without many dust injections; however, on October 15, 1951, while on a train trip he was awakened by an attack of buzzing tinnitus and a blocked sensation in the left ear (see A in Fig. 5). Histamine, 0.1 cc. of 1:100,000,000 dilution gave no relief of symptoms, but following an increase to 0.1 cc. 1:10,000,000 histamine three days later, an immediate improvement began and continued while taking the same histamine dosage twice weekly, as well as two drops sublingually of 1:1,000,000 histamine. In addition a salt-free diet with potassium chloride was followed, and the patient's usual daily thyroid intake was increased from 2½ gr. to 3 gr. The progressive improvement can be followed in subsequent audiograms (see B, C and D in Fig 5).

The next recurrence of Meniere's disease occurred about two years later in September, 1953, coincidental with the development of his usual autumn and winter nasal symptoms. This time the right ear was involved (see A in Fig. 6). The same histamine injection as in the last previous attack was given (1:10,000,000) and immediate improvement in the right ear occurred (see B in Fig. 6), but nasal symptoms persisted. The addition of 0.1 cc. of 1:1,000,000 house dust to the histamine injection was followed by complete clearing of the right ear and nasal symptoms (see C in Fig. 6).

Comment: Whether the long-standing house dust nasal allergy had any causal relationship to the Meniere's disease is not so easy to prove as in the previous food sensitive patient; however, it is interesting that the hydrops attacks all occurred in the fall, coincidental with or following the usual exacerbation in a chronic house dust allergy at that time of the year.

SUMMARY AND CONCLUSIONS.

An attempt has been made to evaluate the results of the small optimum dosage of histamine in the treatment of Meniere's disease by studying 55 new cases seen in one year.

Standard criteria by which to judge results in the medical management of Meniere's disease are sorely needed.

As a rule, if histamine is going to be effective, the relief from symptoms is prompt and definite within hours after injection.

Although only six cases of the 55 (11 per cent) could be shown to be of specific antigen-antibody allergic origin, this possible cause should not be overlooked. In our experience, the specific allergic cases properly managed give the best possible results.

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DEUTSCHE GESELLSCHAFT DER HALS-NASEN-OHRENARZTE.

The 25th Annual Meeting of the Deutche Gesellschaft der Hals-Nasen-Ohrenarzte will be held June 2 and 5, 1954, at Marburg-Lahn. For further information, address Prof N. Mittermeier, Marburg-Lahn, Germany.

SALIVARY GLAND TYPE TUMORS OF THE HEAD AND NECK.*†

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and
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Salivary gland-type tumors occurring in aberrant locations about the head and neck have been of interest ever since Robin[9] described a "mixed tumor" of the palate in 1852, and Forster[3] recognized a similar nasal tumor in 1859. Despite the fact that recently multicentric tumors of this variety were produced in animals by inoculation with a filterable virus, their etiology has never been .satisfactorily explained, and in each theory concerning their nature it is hard to explain all of the different constituents. They are now generally considered to be purely epithelial in origin, although McFarland[6] and some other pathologists do not hold this view. If we accept the epithelial origin, the original term of "mixed" tumor by which they are commonly designated must be discarded. No one type of treatment has found universal acceptance, and in the clinical discussions a wide variation in the type of treatment was noted. End result studies of treatment are infrequent, for the exact assessment of the frequency of recurrence and subsequent outcome is difficult, because of the variable and often long intervals before recurrence.

It has been estimated by Willis[11] that 10 per cent of these tumors occur in locations other than the salivary glands, with approximately half in the palate. In this report of 19 aberrant mixed salivary gland-type tumors, the youngest was 13 years and the oldest 68 years, with an average age of 46. There were nine males and ten females. Six were situated in the palate, two in the nasal septum, two in the sinuses, seven in the pharynx, and one each in the larynx and trachea. While

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nine of the cases exhibited recurrence or extension only two metastasized, and these were to the cervical lymph nodes. Five patients have died, and one is living with tumor (see Table 1).

TABLE I.

Salivary Gland-Type Tumors in Aberrant Locations.

Location	No.	Treatment		Recurrence	Living	Dead
		Surg.	Surg. Irrad.			
Septum	2	1	1	2	1	1
Sinuses	2	1	1	2	1	1
Palate	6	6	0	0	6	0
Pharynx	7	7	0	3	4	3
Larynx	1	1	0	1	1	0
Trachea	1	0	1	1	1	0
Total	19	16	3	9	14	5

Pleomorphism is a prominent feature, and any or all varieties of structure may be found in one tumor. They may resemble simple adenomas on the one hand, or frankly malignant anaplastic carcinomas on the other. On the whole, the growths outside of the salivary glands seem to have a higher degree of malignancy than those in the salivary glands themselves.

Most salivary gland-type tumors occupy a borderline position. They are often classed as benign, but since they have been demonstrated microscopically to be nonencapsulated, recur after removal, widely infiltrate the surrounding tissue, and occasionally metastasize, this designation is not tenable. Likewise, it does not seem accurate to classify them as benign tumors which subsequently become malignant, although Slaughter, Southwick and Walter[10] found 12 out of 212 cases in the parotid gland itself which were thought to have originated from pre-existing benign tumors. They should be considered as malignant from the beginning, although their rate of proliferation and degree of malignancy may sometimes show more or less abrupt increases. The most reasonable concept is to regard them all as low grade malignant adenocarcinomas, regardless of the histologic detail. The question to be answered is not whether the tumor is benign or malignant, but how innocent or malignant is the particular tumor?

Encapsulation of salivary gland-type tumors occurring in aberrant locations is seldom complete. Even when grossly the margin of the tumor appears sharply delimited and encapsulated, microscopic examination will commonly reveal extension into the surrounding area. Because of this the risk of postoperative recurrence is considerable when a sharp line is hewn between the visible tumor and the adjacent tissue. Although the origin of these tumors from multiple foci, with subsequent development of a new tumor, must be considered in recurrent cases, the usual cause for recurrence is conservative or incomplete surgical removal.

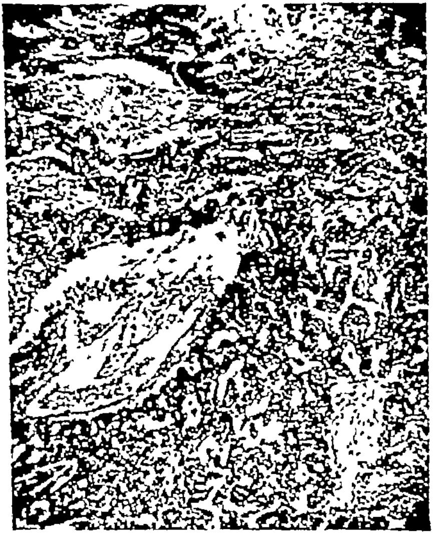

Fig. 1. Histologic picture of salivary gland type tumor of *nose*. The closely packed masses of cells with apparent invasive tendencies was considered malignant, yet the patient remained well for eight years after local excision. Recurrence was treated by excision with a four-year follow-up free from disease.

At present an accurate prediction regarding the outcome of any one tumor cannot be made, and it is questionable whether any salivary gland-type tumor can ever be regarded as completely cured. Certainly the microscopic structure of a salivary tumor is an unreliable guide to prognosis. Recurrence may follow removal of tumors of the most benign-looking histologic type, while cellular active-looking growths, from which recurrence and local invasion was anticipated, may fail to show malignant tendencies. In the following case with involvement of the nasal septum, histologic study revealed masses of cells which appeared to be rapidly growing without limitation by fibrous tissue or a capsule, leading to an initial diagnosis of basal cell carcinoma (see Fig. 1), yet eight years elapsed after local removal of the tumor before a recurrence developed.

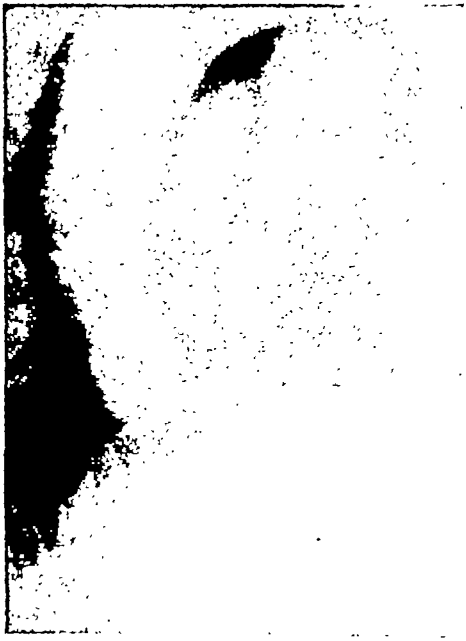

Fig. 2. Firm, discrete, slightly nodular salivary type tumor of cartilaginous and bony septum projecting into right nasal chamber.

Case 1. A 48-year-old male first noticed difficulty in breathing through the right side of the nose in 1934. For the next 10 years polyps were removed from both sides of the nose at frequent intervals. Both the Wassermann and Kahn reactions were strongly positive. In 1944 the blockage on the right became almost complete, and he was admitted to Jefferson Hospital, where a mixed salivary gland-type tumor of the nasal septum was found (see Fig. 2). Surgical excision of the tumor and involved portion of the septum was done.

The patient remained well until 1952, at which time he returned with recurrence of the tumor in the floor of the nose and posterior septum. There was no involvement of the turbinates or sinuses. At operation the remaining septum and the mucosa of the entire floor of the nose was removed through an alveolar approach, and an area of bone destruction in the hard palate approximately ½ cm. in diameter was uncovered. The buccal mucosa was intact over this area, and curettage of the edges of the exposed bone was done without additional bone removal. It would seem likely that the tumor originated in the palate, and extended upward into the nasal septum.

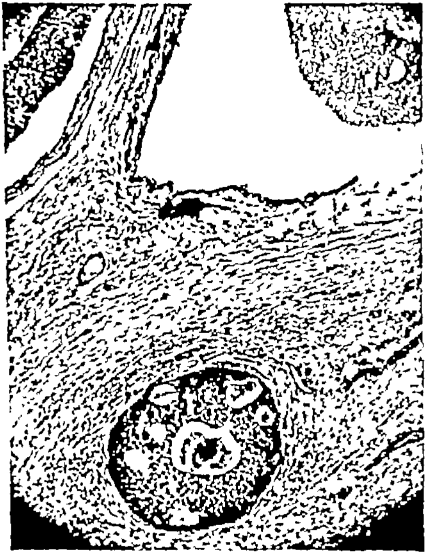

Fig. 3. Histologic section of salivary gland type tumor of antrum. Because of the discrete acini surrounded by large areas of fibrous tissue, with distinct encapsulation the lesion was diagnosed as benign. Despite radical surgical removal the patient died one year later from intracranial extension

To illustrate further the unreliableness of the histologic structure in prognosis and treatment, the histologic appearance of sections taken from a tumor which involved the antrum and lateral nasal wall revealed discrete acini with a large amount of fibrosis, which was thought to be relatively benign and non-invasive (see Fig. 3). This patient died

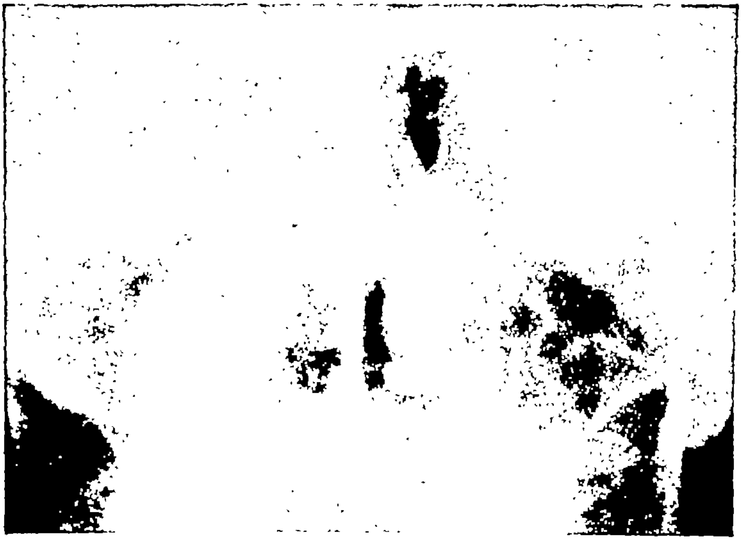

Fig. 4. Roentgenogram with a large, smooth, circumscribed tumor of the medial wall of the right antrum, which proved to be a salivary gland-type tumor.

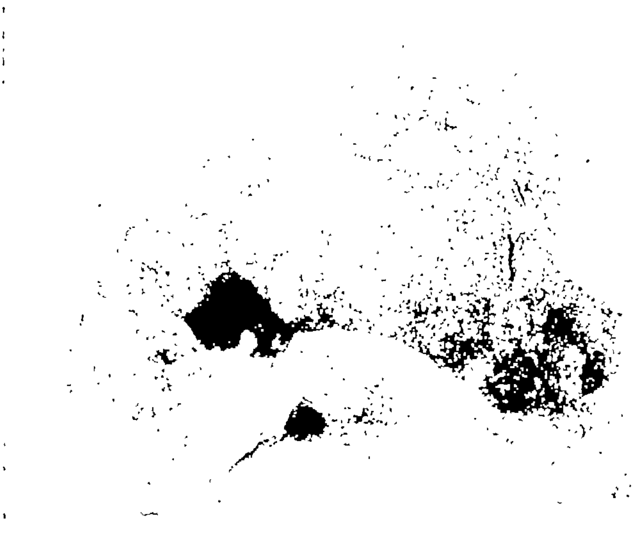

Fig. 5. Specimen of large encapsulated antral salivary tumor with hard palate and excised normal tissue around the periphery of the lesion (inner aspect).

within a year from local extension of the tumor into adjacent intracranial structures

Case 2 Difficulty in breathing through the right side of the nose had been noticed for two months by a 49-year-old white female For a few weeks fullness and tightness along the right side of the nose and occasional blood tinged secretion from that side had been present. She had also observed slight swelling over the right cheek

Examination revealed soft tissue swelling of the right cheek, and narrowing of the right nasal airway due to protrusion medially of the lateral wall. On the roentgenogram (see Fig 4) a density in the floor and medial wall of the right maxillary sinus was observed Tissue was removed intranasally and reported as a salivary gland tumor benign type

At operation through an external approach the tumor was found to extend up to the ethmoids and down to the hard palate The tumor mass was removed as a block which included the hard and soft palates lateral nasal wall and antral floor (see Fig 5) Thorough electrocoagulation of the remaining tissue was performed. The patient was fitted with a prosthesis and discharged from the hospital

Eight months later diplopia developed with recurrence of the tumor in the right ethmoid area There was paralysis of the right Vth and VIth cranial nerves and roentgenograms revealed extension of the destructive process into the sphenoid and base of the skull Irradiation therapy using a tumor dose of 4860r over 26 days was carried out with little improvement Death occurred four months later

The symptoms are largely due to obstruction When the growths reach a large size, pressure symptoms or distortion develops Pain is almost invariably absent. Although in the nasal tumors a feeling of fullness and tightness about the head was described, obstruction to breathing on the involved side, together with some secretion which at times was blood tinged, was the major complaint In the palatal cases the patients complained of difficulty with chewing and swallowing and frequently noticed swelling, but there was no ulceration or bleeding Difficulty in swallowing and eating was prominent when the pharynx was the site of the lesion Hoarseness was the main symptom in the laryngeal case, and obstruction to breathing, especially when lying down, occurred in the tracheal case.

The palatal tumors were not found in the midline but near it on one or the other side Since there are no glands in the anterior part of the hard palate, the common location was in the posterior portion of the hard palate or in the soft palate Although excavation and perforation of the palatal bone was present in three of the palatal cases it did not denote invasion When the tumor was fixed to the underlying bone the resulting crater was due to pressure atrophy These tumors were rounded, well circumscribed, not ulcerated, and could easily be separated from the surrounding tissue. No recurrences have been observed as yet in this group. An illustrative case of a palate tumor follows:

Case 3. A 56-year-old female had a "growth in the throat" for 32 years. The year previous to seeking medical advice and treatment she had observed a gradual increase in the size of the tumor, for which she received "needles" and vitamin therapy from her local physician to shrink the lump.

On anterior rhinoscopy a large tumor mass was visualized on the right side of the nasopharynx arising from the soft palate. When the oral cavity was examined a large, firm, smooth, rounded mass occupying the right half of the soft palate and extending across the midline to the opposite side was noted (see Fig. 6).

When the mass was removed it was found to be limited to the soft palate and faucial region. The pathologic report was mixed tumor.

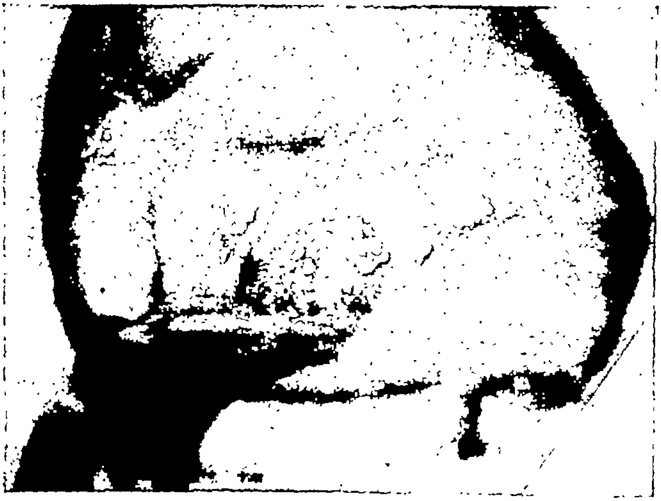

Fig. 6. Appearance of salivary gland tumor of palate. The lesion at the junction of the hard and soft palate on the right side can be seen as a smooth, round mass projecting behind the denture.

In the four cases involving the nose and the sinuses, all developed recurrences. One patient with involvement of the maxillary and ethmoid sinuses died less than one year after the original diagnosis and treatment. Death in this case was due to direct extension of the tumor into the cranial structures, but there was no evidence of regional or distant metastasis. In the other case of a reported benign lesion in the antrum, recurrence occurred after an extensive local removal, and resection of the upper jaw was then required. Only after histologic examination of the entire maxilla was our suspicion confirmed and the diagnosis of a malignant tumor made.

In the seven pharyngeal cases there were three recurrences and three deaths. One of the patients that died developed cervical metastasis. The following patient is living after five years, even though the pathologist considered the tumor malignant and the non-encapsulated mass extended to the base of the skull, so that it was impossible to excise it in one piece, but removal had to be accomplished by morcellation.

Case 4. A 63-year-old white male complained of a sore throat with a feeling of "lump in the throat" for one month. On examination a prominence of the right side of the pharynx occupying the region of the faucial tonsil was observed. The mass was very firm and extended outward to the hard palate, laterally to the mandible and downward to the base of the tonsillar fossa.

At operation the tumor was found to be well encapsulated, extending upward to the base of the skull. As the dissection was performed the upward prolongation broke off and was removed by morcellation. It was not known whether all of the capsule had been removed, but subsequent study failed to reveal any further evidence of the tumor.

Although the pathologic report was malignant mixed tumor of the pharynx, he has had no symptoms and remained free from recurrence for six years.

In the laryngeal case local recurrence occurred in less than two years. Subsequently metastasis to the cervical lymph nodes developed, so that laryngectomy and block dissection of the lymph nodes of the involved side of the neck was necessary.

Case 5. Hoarseness and soreness in the throat of two months' duration were the chief complaints of a 65-year-old male. These symptoms followed a cold, and recently the patient had noticed a sticking sensation in the left side of the neck referred to the left ear.

On examination a marked widening of the vallecula on the left side with displacement of the upper part of the epiglottis posteriorly and crowding of the left aryepiglottic fold to the left, due to a mass, was observed.

Operation was performed through a lateral thyrotomy and the tumor which involved the left ventricular band and pre-epiglottic space was excised. The histologic diagnosis was salivary gland tumor of the larynx. Six months later a recurrence was noted in the left vallecula. He was operated upon through a transhyoid approach and excision of the mass, which involved the pre-epiglottic space extending to the base of the tongue and the lateral pharyngeal wall, was done. The tumor was encapsulated and separated readily from the surrounding tissue. Two years after the second operation the patient returned complaining of blood spitting, and an ulcerating recurrent lesion was found on the posterior surface of the epiglottis. In addition there was an indurated tender area in the left submaxillary region. This time a total laryngectomy with left radical neck dissection and excision of the right submaxillary gland was performed. Histologic examination revealed adenoid cystic carcinoma of the larynx with metastasis to lymph nodes and submaxillary gland.

In the case of the tracheal tumor wide infiltration was observed at the time of operation, so that the entire tumor could not be removed. The patient is living with the tumor, but just how long she will survive before succumbing to the disease is problematical.

Case 6. A 52-year-old female complained of difficulty in breathing, more marked on exertion, and a huskiness of the voice and wheezing at times, of one year's duration. A loss of 17 pounds of weight had occurred.

On indirect and direct laryngoscopic examination there was found a paralysis of the left vocal cord and a large, red mass on the anterior tracheal wall approximately 1½ to 2 cm. below the cord level. The mass almost completely filled the tracheal lumen, leaving an airway of only a few millimeters in the posterior portion. A portion of the tumor was removed, and the histologic diagnoses was mixed tumor of the salivary gland type.

After a tracheotomy had been done the neck was surgically explored, and the tumor was found to involve the larynx and upper trachea with extensive infiltration into the surrounding tissues. The patient received irradiation therapy in the amount of 3500r. to both sides of the neck, with little improvement in the tracheal narrowing or the extent of the tumor. Radical surgical excision is being considered, but as yet has not been done.

Treatment of aberrant salivary gland-type tumors must always be individualized to a certain extent, but the institution of definite therapy as early as possible in every case is fundamental.

To follow pathologic interpretations alone may result in unnecessarily extensive operations, but what is more hazardous they may lead to conservative and incomplete procedures when extensive surgery is needed. The selection of treatment must be based on the coordinated clinical and pathological findings, for the location of the tumor, as well as its histologic structure is a contributing element in determining the extent of therapy. With the possible exception of the palatal tumors the only factor that seems to have any real influence on the final outcome is the radicality of the initial surgery. We can not agree with Dean,[2] who contended that mutilating operations should not be done when dealing with these semi-malignant tumors.

Although the results of surgical treatment have not been encouraging, we believe it remains the best method of treatment. Because these tumors usually have an incomplete capsule, enucleation alone cannot be relied upon as the chief surgical procedure Adequate tissue removal around the periphery of the lesion, including both the soft and necessary bony structures, is the most effective means of preventing recurrence When the extensive surgery always needed to extirpate these tumors in aberrant locations other than the palate is done initially without waiting for recurrence, the recurrence and mortality figures will improve Cervical lymph node metastasis should be treated simultaneously with the primary tumor by radical neck dissection

Since the situation of these tumors in the upper air and food passages, especially when they are located in the ethmoid region, may prevent the ideal surgical procedure of radical removal with the capsule and adjacent normal tissue, the operative procedure described by Hybbinette[4] deserves consideration This surgical approach consists of incision of the capsule with removal of the tumor contents within the capsule After this has been done the capsule is dissected free by sharp and blunt dissection and totally excised This method has merit in the large and bulky tumors in this region, because it allows a careful removal of the capsule after the real tumor is out of the way and the working space is increased One of our cases was treated in this manner because of the large size and extent of the tumor This patient also received preoperative X-ray therapy and implantation of radium seeds, but developed recurrence within a year, and was dead within four years

Certain of the European investigators, notably those in the Scandinavian countries, have a high regard for irradiation therapy Ahlbom[1] thought the best results were obtained with irradiation combined with surgical therapy Pre- and postoperative irradiation was administered in those cases with manifest or suspected malignancy In the clinically benign types no irradiation was given preoperatively but postoperative therapy was used Ohngren[7] expressed a similar view, but advocated electrosurgical treatment. Ringertz[8] utilized both irradiation and electrosurgery, as described by Ohngren

The case for the use of preoperative irradiation has been given as *1* the tumors are made technically easier to remove, *2* prevention of recurrence, and *3* the clinical diagnosis may be mistaken, and the inflammatory lesion will subside under irradiation therapy. It is difficult for us to justfy this procedure, for these tumors have proved so highly resistant to radiation that no real effect has been secured. Of secondary importance is the subsequent fibrosis with which the surgeon has to deal at operation. The high degree of radio-resistance of this group of tumors likewise militates against the success of postoperative irradiation, although on occasion irradiation has seemed beneficial. It is doubtful if its routine use postoperatively has an effect on the prevention of recurrency.

Because of the relative slow growth of this type of tumor and its late metastasis, the mortality should be very low. To date our figures together with those of others, New and Childrey,[6] Ohngren,[7] and Ringertz,[8] have not borne out this optimism (see Table 2). It is questionable whether previous operative trauma has a direct bearing upon recurrence. Failure to appreciate the need for radical surgical procedures would seem to be responsible for the high mortality rate.

TABLE II.

Recurrences and Mortality in Reported Series of Cases.

Author	No. of Cases	Recurrences	Deaths
New and Childrey	48	28	11
Ohngren	11	3	3
Ringertz	37	15	14
Putney and McStravog	19	9	5
Total	115	55	33

SUMMARY.

The slow growth and clinical encapsulation of many of the aberrant salivary gland-type tumors makes us inclined to overlook their potentialities, and influences our surgical judgment. Except for some of the palatal lesions these tumors about the head and neck possess the characteristics of low grade malignancies. Removal of the entire lesion, together with an adequate margin of normal soft and bony tissue utilizing surgical procedures, is preferred over other forms of treatment, but this may not be sufficient to prevent recurrence. In our experience, the addition of irradiation therapy has proved no more beneficial than surgery alone. In a group of 19 cases, six were located in the palate and have remained free from disease Of the remaining 13 cases five have died, two had metastasis and nine developed recurrence.

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OBSERVATIONS ON FRONTAL SINUS DISEASE REQUIRING EXTERNAL SURGERY SINCE 1945.*

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The observations which I am presenting are based upon the clinical and pathological study of 20 cases of frontal sinus disease, which were operated upon externally since 1945. The purpose is to illustrate the clinical manifestations of disease in this sinus during the era of chemo-therapy and bio-therapy, and to present an appraisal of its management.

I have divided the cases into two groups on the basis of nasal symptoms and rhinoscopic findings because these clinical manifestations are commonly considered characteristic of sinus disease. On this basis it was noted that such symptomatic evidence occurred in only six patients, while it was absent in 14. My interest was principally directed to the 14 patients with absent nasal symptoms because they illustrated a wide variety of lesions with diverse clinical manifestations, none of which were referred to the nose.

I believe it is important to emphasize this because too frequently diagnosis and therapy are based on the idea that sinus disease implies nasal symptoms and nasal symptoms imply sinus disease, so I will comment on these patients first. Their many interesting clinical and pathological features warrant a detailed discussion, but as there is not enough time for this I will group them according to similar outstanding clinical features, and will use short extracts from the clinical records and Roentgen findings of one case, to illustrate each group.

The 14 cases can be separated into two main groups, consisting of 10 in which obstruction, partial or complete, of the sinus outlet to the nasal cavity was the important feature, and four in which the primary pathology was outside the sinus and the sinus was secondarily involved. In the 10 cases of obstruction of the sinus outlet the clinical features differed

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because the pathological reaction in the osseus walls was variable. This reaction was one of atrophy due to pressure from an expanding fluid in some, and one of hyperplasia and thickening from inflammation in others. In three of them an external fistula was also present, which entered the sinus through a perforation in its floor.

Six of the 10 cases showed Roentgen findings characteristic of a mucocele, such as absence of scalloping of the sinus wall, increased translucency, and flattening of the medial superior margin of the orbit. In three of these mucocele-like lesions there was an accumulation of a fluid mass in the sinus, with distention of all its walls and marked atrophy of mucous membrane and bone, but there was no sign of any inflammatory reaction or residue. The location of the obstruction varied: in one, it was at a distance from the sinus outlet in the ethmoid capsule, in the other two, it was at the sinus outlet. The cause of the obstruction was obscure in all three cases. A typical example of this lesion is:

J. B., male, age 48, complained of right frontal headache and ptosis of the right eye of three months' duration and gave no history of any

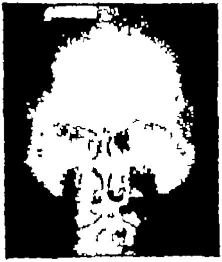

Fig. 1. Roentgenogram of right frontal sinus showing smooth contour, increased translucency, absence of normal densities of sinus walls the result of pressure atrophy of expanding fluid mass retained by blockade of sinus outlet. No inflammatory reaction

sinus infection. He had suffered from a linear fracture through the right parietal and temporal bone at the age of 12 due to a fall, which had caused marked deafness in the right ear, but no connection could be established between it and the present condition. The rhinoscopic examination was entirely negative. He had been seen by an ophthalmologist, fitted with glasses, and referred to a neurologist before an investigation of the sinuses was sought.

Roentgen examination of the right frontal (see Fig. 1) showed typical findings of a mucocele; smooth contour, increased [illegible] bsence of normal densities of the sinus wall. Iodized oil was instilled into the sinus by pushing a needle through the anterior wall, which was as thin as eggshell, and it demonstrated complete blockings of the naso-frontal duct in the ethmoid region. Cure was obtained by simply removing some of the anterior ethmoid cells and relieving the blockade into the nose. The sinus contained heavy grayish mucoid liquid, but no signs of any inflammatory reaction.

Trauma caused a sinus lesion in three patients who showed Roentgen findings of a mucocele without nasal symptoms or rhinoscopic evidence of disease. In them, however, a marked inflammatory reaction was present, with swollen hyperplastic mucous membrane, fibrotic granulation tissue and thickening of the bony walls, instead of atrophy (see Fig. 2). In one of these patients a calcareous mass, due to calcification of a hematoma, had formed in the sinus wall which partly invaded the orbit. There was no evidence of infection in any of them, and the sinus blockade, which caused the mucocele syndrome, was entirely due to inflammatory residue, the result of trauma.

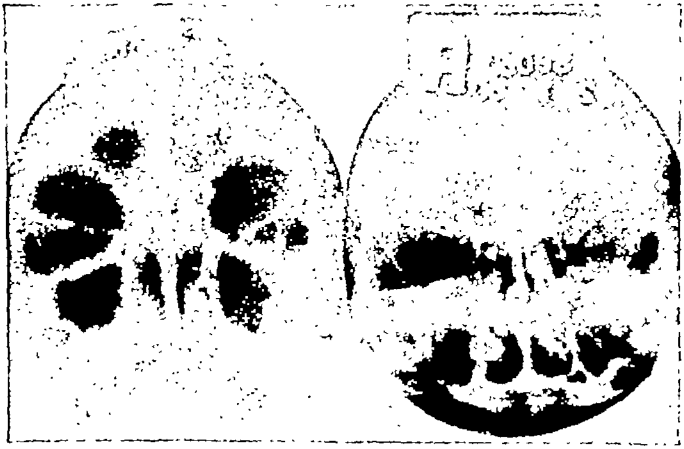

Fig. 2. Roentgenogram of right frontal sinus showing features similar to those in Fig. 1.—there is absence of scalloping, increased translucency, flattening of the medial orbital margin, and in addition increased perisinus density from sclerosis, caused by direct trauma from striking forehead against a door.

Cure was obtained in all three by simply removing the obstructing inflammatory tissue and re-establishing an adequate naso-frontal connection; however, the lesion existed in each case three months to one year before this treatment was carried out. An example is:

A woman, age 59 years, who complained of a tender swelling over the medial aspect of the right orbit and forehead, which developed after striking her head against a door three months prior to admission. It was large enough to interfere with turning her eye medially and upward, and was smooth and compressible like a cyst. The Roentgen findings resembled those of a mucocele and were very similar to the first case (see Fig. 2).

They showed absence of scalloping, increased translucency and flattening of the medial margin of the orbit, but in addition increased perisinus density from sclerosis. External opening of the cystic swelling revealed a brown, thick, pasty material, swollen mucous membrane, and fibrotic granulation tissue which completely blocked the ostium of the sinus. No evidence of infection was present, and culture was negative The clinical manifestations and Roentgen findings were due to pressure from the expanding fluid mass contained within the sinus by the blockade of its outlet with inflammatory residue. This lesion was first diagnosed as a probable dermoid cyst and its origin from the frontal sinus was not suspected until it was incised and the sinus cavity entered.

Mucoceles are commonly reported as specific lesions or disease entities involving nasal sinuses; however, observations from these six patients indicate that they are simply a peculiar physical state of the sinus; namely, an expanding fluid mass confined within the cavity by an obstruction of its outlet, and are only one phase of a disease process, the etiology of which may be varied.

There were four patients with absent nasal symptoms and rhinoscopic findings, whose illnesses began with acute cellulitis over the left frontal sinus and eye. Three of them developed an external fistula at the middle third of the brow, which existed with intermittent drainage for periods of five months, 10 months and 18 months, respectively, before the underlying pathology in the frontal sinus was investigated, and during this time a wide variety of futile therapeutic measures were applied, including hot compresses, sulfas, Roentgen therapy and antibiotics. One of the patients was hospitalized as an emergency, because of a generalized convulsive seizure which was due to localized osteomyelitis of the posterior wall of the sinus and pachymeningitis of the underlying dura. Another was hospitalized because of opacity of the cornea and chronic conjunctivitis due to deformity of the upper lid from scar tissue. Cure was obtained in all by external surgery to remove necrotic tissue, inflammatory debris and establish an adequate naso-frontal connection.

The patient with corneal opacity due to deformity of the upper lid is taken as an example (see Fig. 3). She was a 65-year-old woman who gave a history which made no reference to the nose or sinuses. Four years prior to admission a painless swelling occurred on the left upper lid, which very slowly increased in size for a period of two years, when it rapidly got larger, red and very painful. It was diagnosed as an abscess of the upper lid and was incised at the mid-point of the brow. Pus discharged, relieving the swelling, redness and pain. She was then treated over the following months with sulfas, penicillin and Roentgen therapy, but the discharging fistula persisted, and scar tissue gradually retracted the upper lid so that it was unable to cover the cornea, resulting in opacity, chronic conjunctivitis and blindness.

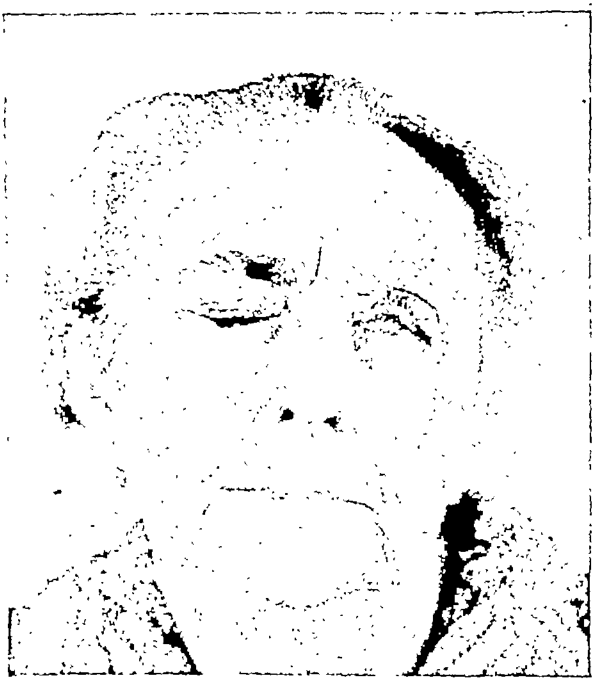

Fig. 3. Patient with fistula into the left frontal sinus of 10 months duration. Contracting scar tissue caused permanent retraction of upper lid with exposure of cornea resulting in opacity and blindness.

The Roentgen films (see Fig. 4), showed clouding of the left frontal sinus, thickening of its walls and marked condensing osteitis surrounding it. External operation revealed a defect in the floor of the sinus measuring 2 x .5 cm., the margin of which was atrophied to knife-like thinness. The sinus was a moderately large single cavity, with markedly thickened intact membrane except at the site of the fistula. The posterior and anterior walls were markedly thickened, and had a pebbled appearance. A probe was passed from the nasal chamber through a large normal anterior ethmoid cell, but was stopped by a thin partition of bone which sealed off the frontal sinus. When this partition was removed the ethmoid cell provided a very suitable naso-frontal duct.

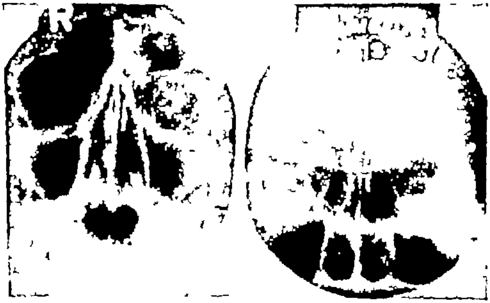

Fig. 4. Roentgenogram of the patient with fistula and corneal opacity, showing clouding of the left frontal sinus, thickening of its walls, and marked condensing osteitis surrounding it.

The biopsy report on the lining membrane of the sinus was pseudo-stratified, ciliated, columnar and transitional epithelium covering edematous vascular tissue which contained plasma cells and lymphocytes. Staphylococcus aureus was reported from the culture. The etiology of this lesion is obscure, and there is nothing in the history or clinical findings to account for the closure of the frontal sinus. Pressure from the contained fluid mass caused atrophy and finally perforation of the thin floor, but it did not expand the cavity where the walls were thickened by condensing osteitis.

These four patients again illustrate the futility of any treatment of closed lesions of the frontal sinus without establishing an adequate naso-frontal connection. Two of them had active syphilis, which was probably a factor in the pathological development of the lesion, but it is interesting to note that although both received large doses of penicillin for treatment of their syphilis the antibiotic had no effect upon the frontal sinus disease, until nasal drainage was provided.

In four other patients with nasal symptoms and rhinoscopic findings so slight as to be of little diagnostic significance, the pathology causing the clinical manifestations proved at operation to be outside of the frontal sinus. In two of these the ethmoid sinuses were involved with an expanding lesion caused by obstruction to the cell outlet, which impinged upon the frontal sinus. The other two were instances of frontal bone cysts encroaching upon the frontal sinus; one an epidermoid, the other a cyst of traumatic origin. Frontal bone cysts are of interest to us because they illustrate that the frontal bone can be the site of cavities other than the frontal sinus, and they must be considered in any careful diagnostic study of frontal sinus disease.

I cite as an example a patient with a cystic lesion due to trauma, which was immediately adjacent to a chronically infected frontal sinus. She was a woman aged 67 years, with a history of prolonged nasal treatment and right middle turbinectomy for nasal discharge. A year prior to admission she developed drooping of the right upper lid, and right frontal headache. Trauma to the right frontal area had occurred from bumping her forehead repeatedly against the frame of the upper bunk of a double deck bed.

Examination showed ptosis of right upper lid and marked limitation of upward movement of the right eye. Some crusting at the site of the turbinectomy was the only nasal finding. Systemic examination revealed diabetes, hypertension and secondary anemia.

Roentgen examination showed a large area of decreased density adjacent to the upper border of the right orbit near the outer canthus measuring 3 cm. in diameter, which was circular and smooth. The frontal sinuses appeared clouded and were not definitely outlined until an iodized oil study was done. This opaque study revealed irregular filling of the frontal sinus due to hyperplastic mucosa, and showed it separated from the round radiolucent area (see Fig. 5).

External opening of the sinus revealed a large lobulated cavity lined by a thickened adherent membrane which was separated from the round cystic area by a bony wall. The supraorbital plate was absent in its lateral half, where a greenish-gray cystic mass encroached upon the orbital space. The cystic cavity was without any epithelial lining and contained granulation tissue intermingled with a glue-like substance. Culture of pus from the frontal sinus showed staph-aureus and non-hemolytic streptococcus, but culture of the material from the cyst showed no growth. This was a dual lesion, a frontal bone cyst resulting from trauma, as described by Geschickter and Copeland,[1] and chronic frontal sinus disease.

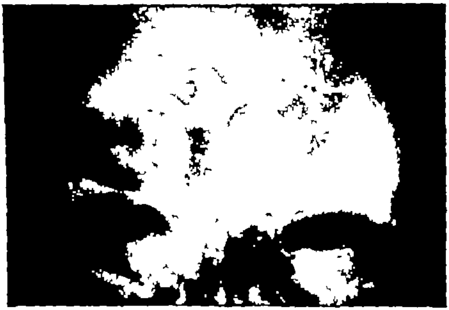

Fig. 5. Roentgenogram of opaque study showing irregular filling of the frontal sinus due to hyperplastic mucosa and its separation from the round radiolucent area which was the site of the traumatic cyst.

Frontal sinus disease manifesting characteristic clinical symptoms and rhinoscopic findings was observed in only six patients, and they all complained of nasal discharge, nasal obstruction and headache or localized pain and tenderness over the frontal sinus. All of them had suppurative infection of the sinus, and all showed characteristic Roentgen findings and clinical manifestations of the disease. The small number of such patients is worthy of comment, because it indicates how greatly chemotherapy and antibiotics have controlled suppurative infection in these sinuses.

My observations on this group are limited to brief comments on two patients: the first, a man age 57, with a history of chronic sinus infection extending over many years. He was subject to acute attacks characterized by frontal headache, purulent nasal discharge and nasal obstruction which were usually relieved with antibotic therapy and local nasal treatment; however, during one attack he developed edema and ptosis of the upper lid, edema of the lower lid, injection of the bulb, lachrymation and marked tenderness at the mid-point of the right brow, in addition to the nasal symptoms. These symptoms persisted and became worse, in spite of antibiotic therapy for a period of two weeks.

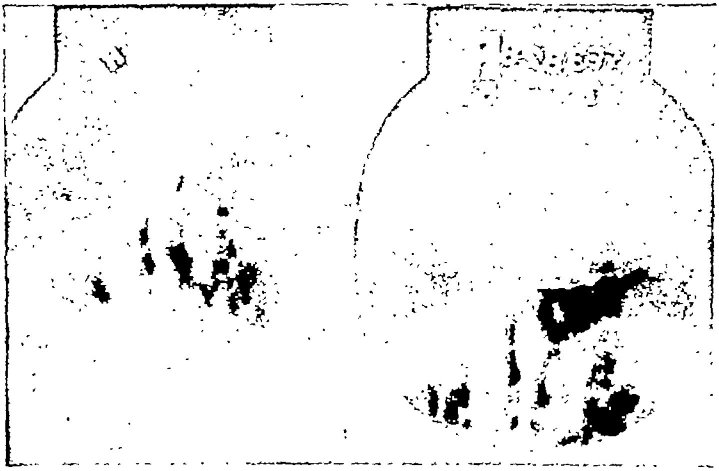

Fig. 6. Roentgenogram showing increased density of right antrum, right ethmoid and both frontal sinuses and in addition the floor of the right frontal thinned out and depressed toward the orbit from an expanding fluid mass within the sinus—a finding characteristic of mucocele.

The Roentgen film showed increased density of the right antrum, right ethmoid and both frontal sinuses. In addition, the bony elements of the floor of the right frontal appeared thinned out and depressed toward the orbit from an expanding fluid mass within the sinus, a finding characteristic of the mucocele-like (see Fig. 6) lesion observed in the first six patients.

External operation revealed a large erosion or perforation of the sinus floor with its muco-periostal lining bulging into the orbit, and when this was incised the cavity of the sinus was found filled with thick cream-like pus. A small probe was passed through the naso-frontal duct from the nasal cavity, showing it to be patulous but partially obstructed by swollen hyperplastic membrane. This obstruction was sufficient to put the pus under pressure, and cause expansion and erosion of the sinus floor, and his cure depended upon its removal regardless of antibiotic therapy.

The second patient, a 22-year-old man with acute fulminating frontal sinus infection, the result of swimming, complicated by staphylococcus bacteremia, was of interest because he illustrated the miraculous results that are possible with chemotherapy and biotherapy supplemented by a simple surgical procedure to promote drainage, in contrast to the futility of all therapeutic measures before the era of chemotherapy and antibiotics. This infection began as an ordinary head cold following swimming, but within 24 hours sudden severe pain occurred in and around the left eye, and he felt very ill. The symptoms increased rapidly, and within 48 hours he had a temperature of 104°, was very toxic, and had severe pain in his right hip. He was admitted to the hospital, and examination revealed acute suppurative infection of the left frontal sinus with pus draining from the naso-frontal duct.

In addition, he had a metastatic infection of the right hip. Roentgen examination confirmed the frontal sinus infection, and blood culture revealed a very heavy growth of staphylococcus aureus, hemolyticus, which was resistant to penicillin, terramycin and streptomycin, but was sensitive to aureomycin. Treatment consisted of aureomycin 500 mg. sixth hour, erythromycin 300 mg. sixth hour, and gantrisin 1 gm. fourth hour. In addition, nasal drainage was supplemented by removing a portion of the sinus floor through a small external incision and inserting a polyethylene tube, through which the sinus was irrigated with normal saline. The sinus infection cleared up within a week, but it required 37 days of chemotherapy and biotherapy to clear the blood stream, during which he took a total of 23,900 mg. of erythromycin, 16,500 mg. aureomycin and 150 mg. gantrisin.

The role of surgery was secondary to that of the chemotherapy and biotherapy in this patient, but I believe it played an important part because it assured free drainage and prevented expanding pressure from the accumulation of a purulent mass within the sinus cavity.

SUMMARY.

These observations indicate that the majority of patients with frontal sinus disease, due to a wide variety of lesions, are free of nasal symptoms and signs; in fact, eye symptoms and ocular findings were much more important in diagnosis than nasal symptoms and rhinoscopic findings. Delay in making an accurate diagnosis and adopting adequate therapy was a notable feature of the management of many of these patients, and they were subjected to futile eye surgery, useless Roentgen therapy, ineffectual biotherapy and chemotherapy, and also incision of overlying soft tissue abscesses which merely served to create discharging fistulas and scar tissue deformities before the true nature of the disease was recognized. Such delay indicates that nasal symptoms are considered by patient and practitioner to be the cardinal evidence of sinus diseases, and when they are absent investigation and treatment are apt to be directed to other fields than the sinuses.

These observations also illustrate that chemotherapy and the antibiotics have altered the course of suppurative infection in the frontal sinus, so that external surgery is required chiefly as a supplementary measure in its treatment; however, there is a primary need for surgery when mechanical obstruction of the sinus outlet is the basis for the pathology and clinical manifestations of the disease, and antibiotics will fail to prevent or cure it without relieving this obstruction. Consequently these two important therapeutic agents should not be considered in any way as rivals, but as being complementary to each other. It is not a question of giving one and then if it fails resorting to the other; instead, adequate therapy requires skill in the use of both.

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1830 Spruce Street.

CORRECTION.

In the November, 1953, issue (Vol. LXIII, No. 11, pp. 1089-1095), page 1092, line 12, the paper by Dr. Felix E. LeBorgne of Ibicuy 1210, Montevideo, Uruguay, reads "originating in the free border." This should read "originating above the free border . . . . ".

BACTERIOLOGY AND VIROLOGY OF ACUTE INFECTIONS OF THE RESPIRATORY TRACT.*†

ALBERT P. MCKEE, M.D.,
(By Invitation).
Iowa City, Iowa.

Much has been said and written about various microbial agents and their relationships to acute respiratory infections. It would seem worthwhile to depart from this rather stereotyped approach occasionally and to weigh certain fundamentals with broad applications. Such is the object of this treatise, and, accordingly, a minimum of space will be devoted to a detailed discussion of any particular genus or species. Conversely fundamental characteristics encompassing many microbial agents will be given proportionately larger consideration.

Microbes exist as populations. This statement is significant whether one deals with a mixed normal flora or an alleged pure culture of infection. While the normal flora of the nose, throat and mouth varies some from one individual to another, the fact remains that it is a mixture of not only different species but also of different genera. The ratio of one species or genus to another may be important. Several instances seem to illustrate the case in point. *Hemophilus influenzae* was considered to be a relatively uncommon component of the normal oral cavity before the use of penicillin allowed it to be cultured in high incidence.[1] *Candida albicans* is found in varying numbers quite frequently in apparently normal mouths.[2] The increase in candida infections may be related to an upset in the balance of the microbial flora although there are other possible explanations.[3] Acute toxic deaths following prolonged antibiotic therapy with penicillin and streptomycin in which staphylococci were present in large numbers

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* Read at the meeting of the Middle Section of the American Laryngological, Rhinological, and Otological Society, Inc., St. Louis, Mo., January 18, 1954.

† From the Department of Bacteriology, The State University of Iowa.

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have been reported.[4] Here again one might suggest that an upset in the total population may have played a part.

While antibiotics have been used to illustrate the promotion of disparity among the total microbial population, other factors may also play a part: the influence of diet, pH, natural microbial antagonism and the immune state may also influence normal flora.

Most studies of microbial populations have concerned bacteria; nevertheless, some contributions relative to viruses lead us to believe that interrelations between viruses and bacteria and between viruses and viruses also exist. Two different types of influenza viruses have been demonstrated to propagate side by side through numerous serial passages in the chick embryo.[5] Competition, among homologous and heterologous viruses, for perhaps the same host cell receptor, has been suggested. That certain viruses can block or partially inhibit the propagation of others in the same locale has been repeatedly demonstrated.[6]

In addition to alterations in total populations a single species is beset with much potential variation. In a very restricted sense one could question whether a pure culture ever exists. In a practical sense it does; however, the inherent adaptability and proneness to mutate or vary among microbes makes one acknowledge that he deals with a very labile progeny, even if they spring from a single cell isolation.

While the incidence of labile cells among the total population may be low, these cells are none the less important. These are the cells that so readily adapt themselves to therapy or other would-be adverse situations. The fact that such individuals may exist in low ratios such as 1:10,000 or greater loses its significance when microbial growth rates are considered. Bacteria such as *Bacillus megatherium* and *Escherichia coli* have been known to reproduce themselves in 9 and 16.5 minutes, respectively.[7,8] Since this multiplication is geometric the uncommon cell today, that is the 1:10,000, literally becomes the common cell tomorrow. Any of the repressive influences listed above may promote this very change.

Many changes among bacteria are dynamic and not at all permanent. The ebb and flow of these changing circumstances suggest an equilibrium propensity on the part of the microbe. Some observers have noted this change in the course of antibiotic therapy. In such instances staphylococci isolated from various infectious processes have increased in resistance from an early 10 per cent resistance to a 60 per cent resistant incidence plateau. When the antibiotic in questions was withdrawn from the therapeutic armamentarium the resistant incidence fell to approximately 40 per cent but returned to 60 per cent again when penicillin was reinstated.[9] The response of bacteria to antibiotics undoubtedly is not so simple as the two extremes noted above. Already a third state has been described among the Neisseriaceae resulting in streptomycin dependent as well as resistant and susceptible strains.[10] Perhaps the more accurate concept would involve a spectrum of resistance or/and susceptibility.

An even more complicated change than just antibiotic resistance has been shown experimentally. *Diplococcus pneumoniae* has been altered antigenically and in resistance. Under the influence of the appropriate desoxyribonucleotidase and antibiotic the organism was changed to a new type by virtue of synthesizing a new capsule and a new state of resistance by changing from a susceptible to a resistant organism.[11] If such broad biological adaptation could occur *in vivo* the significance becomes apparent. This would offer the microbe two avenues of escape.

While viruses present a considerably more difficult group with which to work, some of their potentialities in regard to genetic recombinations have been studied. Viruses of different characteristics propagated simultaneously in the same medium seem to produce progeny possessing characteristics of both original strains.[12] This if it occurs under natural conditions, could be a factor in explaining some of the vagrancies involved in immunizing against influenza. This would not have to be the only influence involved, however. Investigators interested in viruses attacking bacteria have demonstrated a number of factors influencing bacterial virus propagation and have revealed a number of instances of genetice recombinations.[13]

The influence of antibiotics on some of the larger viruses with regard to their growth cycle has provided interesting material for speculation. Members of the Psittacosis-Lymphogranuloma venereum group appear to go through a definite growth cycle.[14] Large initial bodies appear within the cytoplasm of infected cells. These in turn break down to progressively smaller units, the elementary bodies. With cell rupture the elementary bodies are released to parasitize new cells. It is interesting to note that penicillin appears to exert its action early in the cycle, and broad spectrum antibiotics exert their effects later in the cycle.[15] Medium size and small viruses may also have cycles of growth, though this is sometimes difficult to establish. Influenza virus, a medium size virus of about 100 mμ., apparently goes through progressive changes as it is propagated. In the early stages during multiplication it is difficult to demonstrate, so much so that it could be interpreted to exist in a "masked form."[16] Subsequently it becomes more complex, finally to be released from the host cell in the form about which reference is usually made.

The cycle or progressive change in the virus during its propagation does have other than academic interest. The virus may vary in antigenicity, infectiousness and toxicity, and perhaps in other propensities during these various stages. One could readily consider the possibility that this in turn may influence the results of therapy, artificial active immunization and carrier stages. In the light of the work on the psittacosis virus one might consider that other viruses may have a vulnerable point of attack for some growth-inhibiting agent.

If, at a given point in the reproductive cycle, the virus were antigenically satisfactory, that is to say, capable of stimulating a successful immune state and yet lacking in toxic properties, this would constitute an advance in immunization technique. One could raise the question as to which portion of the cycle might most successfully be immunized against. Does the antigenic variation phenomenon stem from some particular part of the cycle? These questions not only justify the research aimed at understanding the method or methods of viral propagation but may in addition yield information of very practical significance.

The masked or immature forms of viruses, if they exist generally, may provide some explanation of the whereabouts of certain viruses in interepidemic periods. Few would question the existence and importance of human carriers of bacterial pathogens. Proving the existence of human carriers of viral pathogens presents a more difficult problem. A number of pathogenic bacteria are known to grow in their specific antiserum. In the presence of specific antiserum the virus seems to be at a disadvantage propagation-wise. A neutralized virus may fix itself to a host cell, but its subsequent multiplication, if adequately neutralized, may well be equivocal. The problem of readily demonstrating human carriers of pathogenic viruses, therefore, becomes a real one.

Recurrent herpes not uncommonly makes its appearance in the same area of the skin or mucocutaneous border. This fact has led many to question whether or not the virus might not be existing in several parasitized cells perhaps protected in this fashion from the effects of body defense, existing, as it were, in a balanced state with the host, wherein the parasite propagates insufficiently to cause clinical symptoms, yet is not completely eradicated from the host. This thesis receives considerable support from a chance observation in which latent herpes encephalitis appeared to be provoked following an hypersensitive reaction. In this instance rabbits receiving insufficient virus to produce clinical encephalitis were used six months subsequently to demonstrate anaphylaxis. Seven days following the anaphylactic experiments the rabbits came down with fatal herpes encephalitis. In this same regard the potentiating effect of histamine was experimentally demonstrated.[17]

Examples of this kind, though not numerous, are of sufficient occurrence to focus attention on the possibility of hypersensitive reactions serving as a percussion cap to explode some infectious diseases. The most likely area of applicability would be among those diseases given to recurrence and perhaps also those in which subclinical infections occurred, establishing a quasi-immune state.

The very interesting situation prevailing in swine influenza leads one to speculate about this same situation. The influenza virus in its masked form appears to reside in the lung worm larva which in turn resides in the earthworm. When swine eat earthworms the lung worm larvae find their way to the lungs of the swine. Unless the swine be provoked by some special method no infection results. In nature this may be a meteorological provocation since swine influenza usually becomes apparent during the first cold spell of the season; nevertheless, the situation has been precipitated following repeated intramuscular inoculations of bacteria.[18] The possibility that an induced hypersensitivity may have played a part in unmasking the viral agent must be considered.

A point of importance to be established is whether an infectious agent such as a virus is necessarily destroyed in the immune individual. Experimental investigation tends to back up the idea that virus may remain alive in the convalescent immune animal for some time. Table I summarizing the results of such an experiment appears below.

TABLE I.—THE RECOVERY OF INFLUENZAL VIRUS, TYPE APR8 FROM CONVALESCENT MICE WITH AND WITHOUT THE USE OF CONCENTRATED, INACTIVE, HOMOLOGOUS VIRUS.

	DAYS AFTER INOCULATION									
	5	6	7	8	9	10	11	12	13	14
Using concentrated inactive virus	+	+	+	+	+	—	—	+	*	+
Using saline	+	+	+	+	+	—	—	—		—

* Omitted to extend the experiment another day.

The concentrated heat inactivated homologous virus is believed to cause a redistribution of the neutralizing antibody. By tying up a large amount of the available antibody it is suggested some of the antibody neutralizing the active virus is dissociated, thus allowing the previously neutralized active virus to become infective. This same situation is believed to be operative even in the hyperimmune animal. Experimentally it is possible to show viable virus present for at least two days longer by the reactivation technique than without it.[19] The above findings suggest interesting possibilities. If the virus is capable of surviving in the convalescent immune or hyperimmune individual for a sufficient length of time, it would be faced with less overwhelming odds as far as antibody is concerned. If the antibody titer dropped low enough, a recurrence of the infection might occur. The reoccurrence of the infection should stimulate a secondary immune response allowing the exaccerbation to be short lived. In this vein of thought one cannot help but see the possible fulfillment in such infections as primary and recurrent herpes. The severity of the primary attack is well known as is the relatively milder episodes in the recurrent disease.

To return to the proposition of convalescent carriers of viral diseases, what situation might prevail here? If the immune carrier discharged neutralized virus to susceptible recipients, would the neutralized virus "take root and grow?" Since they were not capable of inciting clinically obvious disease in the carrier would one expect them to in the recipient? To speculate in this area one cannot afford to overlook the facts concerning virus neutralization. A virus may be underneutralized, just neutralized, or overneutralized with its antibody. An underneutralized virus is infectious, a just neutralized virus is not; but the balance is precarious. The investigator need but to dilute the just neutral mixture and it becomes infective.[20] Enzymatic destruction of a small portion of the antibody or dissociation of the antibody in the presence of a small amount of active, inactive, or underneutralized homologous antigen may upset this balance.[21,22] While antigen-antibody reactions are specific, the fact that the same antigen may be found in widely separated biological situations must not be overlooked.

If a just neutral virus lands in the nonimmune recipient the factor of dilution should shortly become effective, for the gradient here would encourage dissociation, not to mention dilution with body secretions.

There is another fact concerning the interaction of the virus and its specific antibody which is well known but not commonly applied to immunological philosophy. The antibody function involved in the complement-fixation tests is not always the same as that involved in the immune state. This is true also in some other *in vitro* antibody tests; however, *in vitro* antibody assays are usually more convenient than those involving the direct challenge of the experimental animal, and, therefore, they are more commonly employed in immunological studies. The presence of complement-fixing antibodies in the individual's serum usually indicates that he has encountered the infectious agent in question and, therefore, should be better able to deal with it than the unexposed; however, at the moment of exposure, he would not necessarily have sufficient protective antibody titer to prevent onset of the infection. Hence, immunological results, like any other results concerned with the practice of medicine, should be subject to careful examination prior to interpretation.

Arbitrarily and usually for convenience, the hypersensitive and immune states are separated. While this may be justified for the purpose of discussion one could question its general advisability. A case in point is the readiness with which immunity function may be overlooked in antitoxin prophylaxis. When the physician administers horse tetanus or diphtheria antitoxins his concern is usually two-fold; first, he is concerned with rendering the patient passively immune; second, he is concerned with the possibility of his patient's being hypersensitive to horse serum. It is well known that to administer horse serum to the individual hypersensitive to horse protein may result in anaphylactic shock. Even if this is his first exposure, serum sickness may present a problem. The third concern is often overlooked. The individual who has once received horse serum is in a sense immune to it, in that he is capable of eliminating it at a faster rate than someone who has not previously been exposed to this very good foreign protein antigen.[23] This fact becomes of practical significance under any circumstances where additional doses of prophylactic serum seems advisable. Generally speaking, one should think of the hypersensitive and immune states concomitantly lest he stress one aspect to the exclusion of the other.

Finally, close cooperation between physician and microbiologist is not only desirable but also necessary for best results. Such an ideal situation goes far in deciding the significance of laboratory findings. To illustrate this a not uncommon problem may be examined. The diagnosis of pulmonary mycosis usually requires considerable study to substantiate it definitely. The demonstration of sulphur granules in the patient's sputum might lead one to a hasty diagnosis of actinomycosis. The sources of these granules, however, may be chronically infected teeth or tonsils. Repeated rather than an occasional demonstration of the granules and demonstrating them in a specimen obtained by a bronchoscope adds weight to the findings. The exclusion of other possible pathogens such as *Mycobacterium tuberculosis* lends additional weight to the diagnosis. The fact is that some potential pathogens may exist as opportunists among saprophytic normal flora, and the relative incidence of any one organism in a given area may be upset following antibiotic therapy; therefore, laboratory findings should always be weighed carefully in each instance so as to make the most intelligent use of them.

It is difficult to summarize fundamentals for they in themselves are essentially not reducible; however, the philosophical attitude toward the fundamentals referred to above perhaps can be epitomized. Any physician dealing with infections does well to keep in mind at all times that he deals with one of the most versatile antagonists of mankind. Any physician dealing with hypersensitive states must raise his eyes to the horizon of immunology realizing that it is a broad and interrelated field. Last but not least, close cooperation between the basic scientist and practicing physician is mutually beneficial.

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AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

PROGRAM.

Tuesday, May 25, 1954

Time

9:00 A.M. *Business Meeting:*
Election of Candidates.
Election of Nominating Committee.
Induction of Newly Elected Fellows.

9:30 A.M. *Presidential Remarks:*
LeRoy A. Schall, M.D., Boston, Mass.

9:40 A.M. *Introduction of the Guest of Honor:*
J. Parsons Schaeffer, M.D., Professor Emeritus of Anatomy, Jefferson Medical College, Philadelphia, Pa.

SCIENTIFIC SESSION.

9:50 A.M. *1.* Mortalities and Morbidities Following 20,000 Tonsil and Adenoidectomies: George O. Cummings, C.D., Portland, Maine.

10:10 A.M. Open Discussion.

10:20 A.M. *2.* Meniere's Disease—An Unrehearsed Table Discussion (No speeches):
Julius Lempert, M.D., New York, N. Y., (Moderator).
Mr. Terence Cawthorne, London, England.
Kenneth M. Day, M.D., Pittsburgh, Pa.
John R. Lindsay, M.D., Chicago, Ill.
Oral or written questions may be submitted from the floor.

11:00 A.M. *3.* The Study of Ciliated Epithelium with the Electron Microscope: (By Invitation).
Don W. Fawcett, M.D., Boston, Mass., (By Invitation)

11:30 A.M. Open Discussion.

11:40 A.M. 15-MINUTE RECESS.

11:55 A.M. 4. Progressive Oral Gangrene Due to Acatalasemia—Colored Motion Picture:
Shigeo Takahara, M.D., Okayama University Medical School, Japan (By Invitation).

Discussion:
William Dameshake, M.D., Boston, Mass., (By Invitation).

12:25 P.M. 5. The Role of Triangulation Roentgenoscopy as a Method of Guidance in the Removal of Opaque Foreign Bodies Beyond Bronchoscopic Vision:
Alfred A. Dorenbusch, M.D., Charlotte, N. C., (Candidate's Thesis).
No Discussion.

Wednesday, May 26, 1954

Time

9:00 A.M. *"In Memoriam."*

Business Meeting:

Report of the Nominating Committee.

New and Miscellaneous Business.

SCIENTIFIC SESSION.

9:30 A.M. 6. Leverage System of Suspension-Fixation Laryngoscopy (with Motion Picture):
Robert B. Lewy, M.D., Chicago, Ill.

9:50 A.M. Open Discussion.

10:00 A.M. 7. Industrial Noise and Occupational Deafness:
Mr. Harry Nelson, Director of Compensation, Industrial Commission State of Wisconsin, Madison, Wis., (By Invitation).

10:45 A.M. Question Period.

11:00 A.M. 8. The Surgical Treatment of the Atresia Auris Congenita. A Clinical and Histological Report: Professor Luzius Ruedi, Zurich, Switzerland.

11:30 A.M. Discussion:
Howard P. House, M.D., Los Angeles, Calif.

11:40 A.M. 15-MINUTE RECESS.

11:55 A.M. *9.* Headache Mechanisms:
Harold G. Wolff, M.D., New York, N. Y., (By Invitation).
No Discussion.

Thursday, May 27, 1954

Time
9:00 A.M. *Business Meeting:*
Election of Council.
Five-Minute Council Meeting.
Presentation of New Instruments.

SCIENTIFIC SESSION.

9:30 A.M. *10.* An Experimental Study of the Small Blood Vessel of the Spiral Ligament and Stria Vascularis of Living Guinea Pigs During Anaphylaxis:
Francis L. Weille, M.D., Boston, Mass.

9:50 A.M. Open Discussion.

10:00 A.M. *11.* A Half Century of Triological Transactions:
Lyman G. Richards, M.D., Wellesley Hills, Mass.
No Discussion.

10:30 A.M. *12.* The Emotional Preparation of Children for Surgery: Television Kinescope:
No Discussion.

11:00 A.M. 15-MINUTE RECESS.

11:15 A.M. *13.* Neoplasms of the Middle Ear and Mastoid:
Wesley H. Bradley, M.D., Syracuse, N. Y., (By Invitation).
James H. Maxwell, M.D., Ann Arbor, Mich.

11:35 A.M. Discussion:
Daniel Miller, M.D., Boston, Mass.

11:45 A.M. *14.* Benign Malignant Lesions of the Larynx—A Motion Picture:
Gabriel Tucker, M.D., Philadelphia, Pa.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

April 1, 1954.

Acousticon Models A-17, A-180 and A-185.
Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 1, New York.

Auditone Models 11 and 15.
Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67 and 70.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac Model M; Mono-Pac Model "Lyric"; Mono-Pac Model "Rhapsody."
Manufacturer: Beltone Hearing Aid Co., 2900 West 36th St., Chicago 32, Ill.

Cleartone Model 500; Model 700; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Model D-1; Dahlberg Junior Model D-2; Dahlberg Model D-3 Tru-Sonic; Dahlberg Model D-4 Tru-Sonic.
Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Fortiphone Models 19-LR; 20A; 21-C and 22.
Manufacturer: Fortiphone Limited, Fortiphone House, 247 Regent St., London W. 1, England.
Distributor: Anton Heilman, 75 Madison Ave., New York 16, N. Y.

Gem Hearing Aid Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.
Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.
Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico Model J; Maico Top Secret Model L; Maico Maxitone.
Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis, Minn.

Micronic Model 303; Micronic Model "Mercury"; Micronic Star Model.
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Classic Model T9; Microtone Model T10; Microtone Model T612.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 106 So. 7th St., Philadelphia 6, Pa.

Normatone Model C and Model D-53.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.

Distributor: Normatone Hearing Aid Co., 22 East 7th St., St. Paul (1), Minn.

Otarion Models B-15 and B-30; Otarion Models F-1, and F-3; Otarion Model G-3; Otarion Model H-1; Custom "5."

Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40. Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny-Myte); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Model 62 Starlet; Model 72; Model 82 (Zephyr).

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Distributor: Radioear Corp., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Silvertone Model H-16, J-92; Silvertone Model P-15.

Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.

Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966; Sonotone Model 977; Sonotone Model 988.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Televox Model E.

Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 953; Telex Model 1700.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.

Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster; Model Cameo.

Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2.

Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal; Zenith "Regent."

Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices have vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TRANSISTOR HEARING AIDS ACCEPTED.

Acousticon Model A300; 1 transistor, 2 tubes. Model A-310; 1 transistor, 2 tubes. Model A-330; 3 transistors.

Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 35, New York.

Audivox, Model 71; 3 transistors.

Manufacturer: Audivox, Inc., 123 Worcester St., Boston 18, Mass.

Beltone Concerto Model; 3 transistors.

Manufacturer: Beltone Hearing Aid Co., 2900 W. 36th St., Chicago 32. Illinois.

Maico Transist-Ear, Model O; 3 transistors.

Manufacturer: The Maico Company, Inc., 21 N. 3rd St., Minneapolis 1, Minnesota.

Micronic "All American" Hearing Aid; 3 transistors.

Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Model T1 (Red Dot); 3 transistors. Microtone Model T1 (Yellow Dot); 3 transistors.

Manufacturer: The Microtone Corporation, Ford Parkway on the Mississippi, St. Paul 1, Minn.

Otarion Model C-15; 1 transistor, 2 tubes. Otarian Model F-22; 1 transistor, 2 tubes.

Manufacturer: Otarion, Inc., 4757 N. Ravenswood Ave., Chicago 40, Ill.

Radioear Model 820; 3 transistors.

Manufacturer: E. A. Myers & Sons, Inc., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Silvertone Model H-25; 3 transistors.

Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.
Distributor: Sears, Roebuck & Co., 925 South Homan Ave., Chicago 7, Illinois.

Sonotone Model 1010; 1 transistor, 2 tubes.

Manufacturer: Sonotone Corporation, Elmsford, N. Y.

Telex Model 954; 1 transistor, 2 tubes.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Unex Model TR-3D; 3 transistors.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Zenith Model Royal-T; 3 transistors. Zenith Model Super Royal-T; 3 transistors.

Manufacturer: Zenith Radio Corp., 5801 W. Dickens Ave., Chicago 39, Illinois.

SEMI PORTABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).

Manufacturer: A. M. Brooks Co., 1222 W. Washington Blvd., Los Angeles 7, Calif.

Aurex (Semi-Portable).

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.

Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. Frederick T. Hill, Professional Bldg., Waterville, Me.
Vice-President: Dr. D. E. Staunton Wishart, 170 St. George St., Toronto 5, Ontario, Canada.
Secretary: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Ill.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Statler Hotel, Boston, Mass., May 23-24, 1954.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Gordon F. Harkness, Davenport, Iowa.
First Vice-President: Claude C. Cody, Houston, Tex.
Second Vice-President: Daniel S. Cunning, New York, N. Y.
Secretary: Harry P. Schenck, Philadelphia, Pa.
Treasurer: Fred W. Dixon, Cleveland, Ohio.
Meeting: Statler Hotel, Boston, Mass., afternoon, May 27; all day, May 28.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. LeRoy A. Schall, 243 Charles St., Boston, Mass.
President-Elect: Dr. Kenneth M. Day, 121 University Pl., Pittsburgh, Pa.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Meeting: Statler Hotel, Boston, Mass., May 25-27, 1954. (Mornings only.)

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. Dean Lierle, Iowa City, Iowa.
Vice-Chairman: Dr. Fred W. Dixon, Rose Bldg., Cleveland, Ohio.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3, Tenn
Meeting: San Francisco, Calif., June 21-25, 1954.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Walter H. Theobald, 307 N. Michigan Ave., Chicago 11, Ill.
President-Elect: Dr. Algernon B. Reese, 73 East 71st St., New York 21, N. Y.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Waldorf-Astoria, New York City, Sept. 19-24, 1954.

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Statler Hotel, Boston, Mass., May 17-22, 1954.
Waldorf-Astoria, New York City, Sept., 1954.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Edwin N. Broyles, 1100 No. Charles St., Baltimore 1, Md.
Secretary: Dr. F. Johnson Putney, 255 So. 17th St., Philadelphia (3) Pa.
Meeting: Statler Hotel, Boston, Mass. (Afternoons) May 25-26, 1954.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. Victor Alfaro.
Vice-Chairman: Dr. Irvin Feldman.
Secretary: Dr. Frasier Williams.
Treasurer: Dr. John Louzan.
Meetings are held on the third Tuesday of October, November, March and May, 7:00 P.M.
Place: Army and Navy Club, Washington, D. C.

THE LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. W. L. Hughes, Lamar Life Bldg., Jackson, Miss.
Vice-President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

OTOSCLEROSIS STUDY GROUP.

President: Theo. E. Walsh, 640 So. Kingshighway, St. Louis 10, Mo.
Secretary: Dr. Lawrence R. Boies, Med. Arts Bldg., Minneapolis 2, Minn.
Meeting: Waldorf-Astoria, New York City, Sept., 1954.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. Albert D. Ruedemann, 1633 David Whitney Bldg., Detroit 26, Mich.
President-Elect: Dr. F. Lambert McGannon, 14900 Detroit Ave., Lakewood 9, Ohio.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2, N. Y.
Meeting: Waldorf-Astoria, New York City, September, 1954.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. J. M. Tato, Azcuenaga 235, Buenos Aires, Argentina.
Executive Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U. S. A.
Meeting: Fifth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
President: Dr. J. H. Font, Medical Arts Bldg., San Juan, P. R.
Time and Place: 1956, Puerto Rico.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Norris J. Heckel, Chicago, Ill.
President-Elect: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.
Meeting: Chicago, Ill., Sept. 22-24, 1954.

THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Peter N Pastore, Richmond, Va.
President-Elect: Dr. G. S. Fitz-Hugh, Charlottesville, Va.
Vice-President: Dr. H. L. Mitchell, Lexington, Va.
Secretary-Treasurer: Dr. L. B. Sheppard, 301 Medical Arts Bldg., Richmond, Va.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Harold Owens, M.D.
Secretary-Treasurer: Robert A. Norene, M.D.

Chairman of Section on Ophthalmology: Sol Rome, M.D.
Secretary of Section on Ophthalmology: Wendell C. Irvine, M.D.

Chairman of Section on Otolaryngology: Max E. Pohlman, M.D.
Secretary of Section on Otolaryngology: Herschel H. Burston, M.D.

Place: Los Angeles County Medical Association Building, 1925 Wilshire Boulevard, Los Angeles 57, Calif.
Time: 6:00 P.M., first Thursday of each month from September to June inclusive—Ophthalmology Section. 6:00 P.M., fourth Monday of each month from September to June inclusive—Otolaryngology Section.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Harry Nievert, 555 Park Ave., New York (21), N. Y.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York (16), N. Y.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. Cecil Swann, Asheville, N. C.
Secretary and Treasurer: Dr. Geo. B. Ferguson, Durham, N. Car.
Meeting: Joint, with South Carolina Society of Ophthalmology and Otolaryngology, Durham, N. C., Nov. 4-6, 1954.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. David S. Asbill, Columbia, S. Car.
Vice-President: Dr. John McLean, Greenville, S. Car.
Secretary-Treasurer: Dr. Roderick Macdonald, Rock Hill, S. Car.
Meeting: Joint, with North Carolina Eye, Ear, Nose and Throat Society, Durham, N. C., Nov. 4-6, 1954.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.
Secretary-Treasurer: Dr. John F. Tolan, 3419 47th Ave., Seattle (5), Wash.
Meeting: Honolulu, 1954.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Edwin N. Broyles, 1100 No. Charles St., Baltimore 1, Md.
Secretary: Dr. F. Johnson Putney, 255 So. 17th St., Philadelphia (3) Pa.
Meeting: Statler Hotel, Boston, Mass. (Afternoons) May 25-26, 1954.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

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Vice-Chairman: Dr. Irvin Feldman.
Secretary: Dr. Frasier Williams.
Treasurer: Dr. John Louzan.
Meetings are held on the third Tuesday of October, November, March and May, 7:00 P.M.
Place: Army and Navy Club, Washington, D. C.

THE LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. W. L. Hughes, Lamar Life Bldg., Jackson, Miss.
Vice-President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

OTOSCLEROSIS STUDY GROUP.

President: Theo. E. Walsh, 640 So. Kingshighway, St. Louis 10, Mo.
Secretary: Dr. Lawrence R. Boies, Med. Arts Bldg., Minneapolis 2, Minn.
Meeting: Waldorf-Astoria, New York City, Sept., 1954.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. Albert D. Ruedemann, 1633 David Whitney Bldg., Detroit 26, Mich.
President-Elect: Dr. F. Lambert McGannon, 14900 Detroit Ave., Lakewood 9, Ohio.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2, N. Y.
Meeting: Waldorf-Astoria, New York City, September, 1954.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. J. M. Tato, Azcuenaga 235, Buenos Aires, Argentina.
Executive Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U. S. A.
Meeting: Fifth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
President: Dr. J. H. Font, Medical Arts Bldg., San Juan, P. R.
Time and Place: 1956, Puerto Rico.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Norris J. Heckel, Chicago, Ill.
President-Elect: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.
Meeting: Chicago, Ill., Sept. 22-24, 1954.

THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Peter N Pastore, Richmond, Va.
President-Elect: Dr. G. S. Fitz-Hugh, Charlottesville, Va.
Vice-President: Dr. H. L. Mitchell, Lexington, Va.
Secretary-Treasurer: Dr. L B. Sheppard, 301 Medical Arts Bldg., Richmond, Va.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President:Harold Owens, M.D.
Secretary-Treasurer: Robert A. Norene, M.D.
Chairman of Section on Ophthalmology: Sol Rome, M.D.
Secretary of Section on Ophthalmology: Wendell C. Irvine, M.D.
Chairman of Section on Otolaryngology: Max E. Pohlman, M.D.
Secretary of Section on Otolaryngology: Herschel H. Burston, M.D.
Place: Los Angeles County Medical Association Building, 1925 Wilshire Boulevard, Los Angeles 57, Calif.
Time: 6:00 P.M., first Thursday of each month from September to June inclusive—Ophthalmology Section. 6:00 P.M., fourth Monday of each month from September to June inclusive—Otolaryngology Section.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Harry Nievert, 555 Park Ave., New York (21), N. Y.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York (16), N. Y.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. Cecil Swann, Asheville, N. C.
Secretary and Treasurer: Dr. Geo. B. Ferguson, Durham, N. Car.
Meeting: Joint, with South Carolina Society of Ophthalmology and Otolaryngology, Durham, N. C., Nov. 4-6, 1954.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. David S. Asbill, Columbia, S. Car.
Vice-President: Dr. John McLean, Greenville, S. Car.
Secretary-Treasurer: Dr. Roderick Macdonald, Rock Hill, S. Car.
Meeting: Joint, with North Carolina Eye, Ear, Nose and Throat Society, Durham, N. C., Nov. 4-6, 1954.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.
Secretary-Treasurer: Dr. John F. Tolan, 3419 47th Ave., Seattle (5), Wash.
Meeting: Honolulu, 1954.

THE RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Chairman: Dr. Isaac H. Jones, 635 S. Westlake, Los Angeles, Calif.
Treasurer: Dr. Pierre Violé, 1930 Wilshire Blvd., Los Angeles, Calif.
Program Chairmen:
  Otolaryngology: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.
  Ophthalmology: Dr. Harold F. Whalman, 727 W. 7th St., Los Angeles, Calif.

Mid-Winter Clinical Convention annually the last two weeks in January at Los Angeles, Calif.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Chas. C. Grace, 145 King St., St. Augustine, Fla.
President-Elect: Dr. Jos. W. Taylor, 706 Franklin St., Tampa, Fla.
Secretary-Treasurer: Dr. Carl S. McLemore, 1217 Kuhl Ave., Orlando, Fla.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President: Dr. Harry P. Schenck.
Vice-President: Dr. William J. Hitschler.
Treasurer: Dr. Chevalier L. Jackson.
Secretary: Dr. John J. O'Keefe.
Historian: Dr. Herman B. Cohen.
Executive Committee: Dr. M. Valentine Miller, Dr. Charles E. Towson, Dr. Thomas F. Furlong, Dr. Benjamin H. Shuster, ex-officio.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Vice-Chairman: Dr. K. W. Cosgrove, 111 E. Capitol Ave., Little Rock, Ark.
Secretary: Dr. F. A. Holden, Medical Arts Bldg., Baltimore, Md.
Meeting:

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James K. Stewart, Wheeling, W. Va.
Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. C. Otrich, Belleville, Ill.
President-Elect: Dr. Phil R. McGrath, Peoria, Ill.
Secretary-Treasurer: Dr. Alfred G. Schultz, Jacksonville, Ill.

CANADIAN OTOLARYNGOLOGICAL SOCIETY
SOCIETE CANADIENNE D'OTOLARYNGOLOGIE

President: Dr. Robert Black, 401 Medical Arts Bldg., Winnipeg, Manitoba.
Secretary: Dr. W. Ross Wright, 361 Regent St., Fredericton, N. B.
Place: Harrison Hot Springs Spa, Harrison Hot Springs, B. C.
Time: June 13-15, 1954.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Oscar Marchman, Jr., Dallas, Texas.
Secretary-Treasurer: Dr. Morris F. Waldman, Dallas, Texas.

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDICO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Salvador Mixco Pinto.
Secretary: Dr. Daniel Alfredo Alfaro.
Treasurer: Dr. Antonio Pineda M.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

President: Dr. Cesar LaBolde, Mexico, D. F.
Vice-President: Dr. M. Gonzalez Ulloa, Mexico, D. F.
Secretary: Dr. Juan de Dios Peza, Mexico, D. F.

FEDERACION ARGENTINA, DE SOCIEDADES DE OTORRINOLARINGOLOGIA.

Secretario del Exterior: Dr. Juan Manuel Tato.
Sub-Secretario del Exterior: Dr. Oreste E. Bergaglio.
Secretario del Interior: Dr. Eduardo Casterán.
Sub-Secretario del Interior: Dr.Atilio Viale del Carril.
Secretario Tesorero: Dr. Vicente Carri.
Sub-Secretario Tesorero: Dr. José D. Suberviola.

ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

Presidente: Dr. J. Abello.
Vice-Presdente: Dr. Luis Suñe Medan.
Secretario: Dr. Jorge Perelló, 319 Provenza, Barcelona.
Vice-Secretario: Dr. A. Pinart.
Vocal: Dr. J. M. Ferrando.

SOCIEDAD NACIONAL DE CIRUGIA OF CUBA.

Presidente: Dr. Reinaldo de Villiers.
Vicepresidente: Dr. César Cabrera Calderin.
Secretario: Dr. José Xirau.
Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Andre Soulas, Paris, France.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa. U. S. A.
Meeting: 3rd International Congress of Broncho-Esophagology.
Time and Place: September or October, 1954, Lisbon, Portugal.

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER— CAMPINAS.

President: Dr. Heitor Nascimento.
First Secretary: Dr. Roberto Barbosa.
Second Secretary: Dr. Roberto Franco do Amaral.
Librarian-Treasurer: Dr. Leoncio de Souza Queiroz.
Editors for the Archives of the Society: Dr. Guedes de Melo Filho, Dr. Penido Burnier and Dr. Gabriel Porto.

SOCIEDAD CUBANA DE OTO-LARINGOLOGIA.

President: Dr. Reinaldo de Villiers.
Vice-President: Dr. Jorge de Cárdenas.
Secretary: Dr. Pablo Hernandez.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA.

Presidente: Dr. Aldo Remorino.
Vice-Presidente: Dr. Luis E. Olsen.
Secretario: Dr. Eugenio Romero Diaz.
Tesorero: Dr. Juan Manuel Pradales.
Vocales: Dr. Osvaldo Suárez, Dr. Nondier Asis R., Dr. Jorge Bergallo Yofre.

BUENOS AIRES CLUB OTORINOLARINGOLOGICO.

Presidente: Dr. K. Segre.
Vice-Presidente: Dr. A. P. Belou.
Secretario: Dr. S. A. Aranz.
Pro-Secretario: Dr. J. M. Tato.
Tesorero: Dr. F. Games.
Pro-Tesorero: Dr. J. A. Bello.

SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLOMBIA).

Presidente: Dr. Alfonso Tribin P.
Secretario: Dr. Felix E. Lozano.
Tesorero: Dr. Mario Arenas A.

SOCIEDAD ESPANOLA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. D. Adolfo Hinojar Pons.
Vice-Presidente: Dr. D. Jose Perez Mateos.
Secretario General: Dr. D. Francisco Marañés.
Tesorero: Dr. D. Ernesto Alonso Ferrer.

ASOCIACION DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DE GUATEMALA

Presidente: Dr. Julio Quevedo, 15 Calle Oriente No. 5.
First Vice-Presidente: Dr. Héctor Cruz, 3a Avenida Sur No. 72.
Second Vice-Presidente: Dr. José Luis Escamilla, 5a Calle Poniente No. 48.
Secretario-Tesorero: Dr. Horace Polanco, 13 Calle Poniente No. 9-D.

FIRST CENTRAL AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY.

President: Dr. Victor M. Noubleau, San Salvador.
Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

SOCIEDAD DE ESTUDIOS CLINICOS DE LA HABANA

Presidente: Dr. Frank Canosa Lorenzo.
Vice-Presidente: Dr. Julio Sanguily.
Secretario: Dr. Juan Portuondo de Castro.
Tesorero: Dr. Luis Ortega Verdes.

FOURTH LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Dario.
Secretary:
Meeting: Lima, Peru, 1957.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY

President: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis, Mo.
General Secretary: Dr. Paul Holinger, 700 No. Michigan Ave., Chicago (11), Ill.
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COCHLEAR MICROPHONIC MEASUREMENTS IN EXPERIMENTAL LABYRINTHINE OCCLUSION AND FENESTRATION.[*][†][‡]

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In the development and perfection of the fenestration operation for clinical otosclerosis, Dr. Julius Lempert has done far more than make available practical serviceable hearing to uncounted thousands of people with hearing losses. He has directed the energies and attentions of research scientists and otologists to the numerous problems in physiology which confront every student of ear function. Paradoxically, the demonstration in otosclerosis of increased sound transmission following fenestration of the horizontal semicircular canal has pointed to many unanswered questions in tympanic and cochlear physiology.

EARLY SURGICAL STEPS IN OTOPHYSIOLOGY.

In 1876 Kessel[1] mobilized the stapes in a case of adhesive otitis with conduction deafness by removing adhesions from the niche of the oval window and cutting the tendon of the stapedius muscle. A temporary improvement in hearing was short lived.

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[*] Read at the meeting of the Western Section of the American Laryngological, Rhinological, and Otological Society, Inc., Portland, Ore., February 6, 1954.

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Passow[2] fenestrated the promontory in 1892 with questionable results. In the same year both Blake[3a] and Jack[3b] reported stapes extraction attempts. Both reported improvement in hearing of short duration, frequently accompanied by surgical complications. Because of disappointing end-results the procedure was dropped, due to the fact that it was frequently impossible to remove the footplate. The pathology of otosclerotic stapedial fixation was poorly understood at that time.

Barany[4] fenestrated the posterior vertical semicircular canal in 1910, apparently in order to mobilize the perilymph without opening the mastoid antrum. In 1913 Jenkins[5] stated that perilymphatic fenestration altered the fluid contents of the perilymph space and thus improved the hearing.

Holmgren reported in 1923[13] that sterile technique permitted safe opening of the labyrinth. He made a fenestra in the anterior vertical semicircular canal, and stated that fenestration restored hearing by relieving excess perilymphatic fluid pressure.

In Sourdille's[6] 1931 technique the fenestration was a component of "tympano-labyrintho-pexie." He stated that the improvement in hearing was due to the freeing of drum membrane from adhesions, thus allowing it to transmit transtympanic air-conducted sound to the round window, which was now able to function as the sensitive portion of the cochlear perilymph. He later advanced the theory of "the mechanism of the half-filled flask," in which the removal of some perliymph left a half empty perilymph space with greater mobility of perilymph.

In 1938 Dr. Julius Lempert published the first of a series of reports on his one-stage endaural fenestration technique which has been adopted as the present standard operation throughout the world.

More recently, Popper[7] stated in support of his modifications that the aim of fenestration is to create a new escape valve, inasmuch as he feels that under normal conditions sound enters through the round window, with the normal oval window acting as an escape valve. In his conception the ossicular chain is purely a damping mechanism to protect the organ of Corti from traumatic vibratory stimuli.

Lempert[9] and his colleagues, in monkey experiments, showed that the improvement in hearing following fenestration of the vestibular labyrinth is not a result of the reduction of fluid pressure.

TYMPANO-COCHLEAR PHYSIOLOGY.

The Helmholtz theory of sound transmission stated that sound vibrations upon the tympanic membrane are transmitted to the oval window via the transformer action of the tympanic ossicular chain. The need for a transformer action is due to the difference in the density of air and perilymph which involves a large change in impedance; without the transformer action there would be a predictable 25 to 30 db loss because of these differences. The lever mechanism of the ossicular chain is an impedance matching device to transform the high amplitude, low intensity air borne sound waves into low amplitude, high intensity fluid borne sound waves. According to this theory, the round window serves as an outlet or escape valve for the perilymph motion within the cochlear capsule.

It is true that some sound does impinge on the round window and depending upon the phase relation, either aids or opposes the sound waves entering through the oval window. The sound pressure differences existing between round and oval window, and between round window and the fenestra location, have been explored and reported by Bekesy.[8] The primary portal of entry remains, and is now conceded to be the oval window under normal conditions, although it is probably true that the round window is a portal of entry under some pathologic conditions. Under those circumstances the basilar membrane yields to stimuli originating in the perilymph of the scala tympani.

Blocking experiments of the round window by several investigators have yielded conflicting conclusions.[10,11] Hughson and Crowe[10] advocated that the round window be blocked as a surgical technique for improvement of hearing. Their report of threshold improvement was probably due to incomplete blocking which may improve hearing since the round window membrane will be shielded from extraneous tympanic sound pressures and thus diminish the phase cancellation; while the presence of a small air bubble, entrapped between the membrane and the blocking material, may well improve the high frequency response. In our estimation these factors, together with the individual idiosyncrasies that must be expected, can account for the conflicting conclusions noted.

The status of bone conduction thresholds in estimation of cochlear neural integrity is open to question. Insufficient clinicopathologic information is available for study.

In our attempt to obtain data on these subjects experimentally, a number of procedures have been performed on cats, utilizing cochlear microphonic potentials (Wever-Bray effect)[15] as a measure of response to both air and bone conducted sounds. These response characteristics, which have been made at acoustic levels well above threshold, have been analyzed under varying conditions of experimental labyrinthine pathologic states.

EXPERIMENTAL SURGICAL TECHNIQUE.

The adult cat is anesthetized lightly with intra-peritoneal nembutal. A lateral cervical approach is utilized. A post-auricular Y-shaped incision is made through skin, superficial and deep fasciae. The underlying sterno-mastoid and trapezius muscles are reflected to expose the tympanic bulla and cartilaginous external auditory canal. The lateral bony wall of the bulla is removed to expose the round window niche and promontory. A platinum electrode is placed on the promontory close to the round window. The indifferent electrode is attached to an exposed neck muscle.

The lamb[illegible]dal ridge is removed to expose adequately the stapes in [illegible] ero-medial "compartment" of the tympanic ity for [illegible] s of fixation, with dental cement. Occa- ally i[illegible] ary to section the descending portion of acial [illegible] his exposure.

[illegible] uce air-conducted sound and eliminate [illegible] ctronic "cross-talk," a plastic tube 35 cm. long, leading from an air conduction hearing receiver, is cemented into the surgically sectioned external auditory canal.

In order to introduce bone-conducted sound, a shielded bone conduction hearing aid receiver is sutured into position on the squamosal periosteum after preliminary reflection of the temporalis muscle. The muscle is resutured over the receiver to maintain it in position.

The fenestrations referred to have all been performed in the external semicircular canal. The fenestra has been left open in these studies, and has not been covered by any membrane. Comparative tests with the bulla open or closed revealed no significant differences.

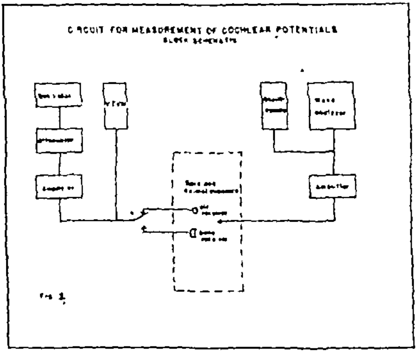

Fig. 1.

ELECTRONIC EQUIPMENT.

The electronic equipment used to measure cochlear potentials is shown as a block schematic in Fig. 1. In effect, a constant voltage is maintained across either the bone conduction or air conduction receiver at all the test frequencies; the resultant *cochlear microphonic potentials* being picked up by an electrode on the promontory near the round window, amplified and measured by a General Radio wave analyzer used as a frequency selective vacuum tube voltmeter. The signal is also displayed on the oscilloscope as a visual check on performance.

In practice the voltage at the receiver can not be kept constant, since either the receiver or the cochlea may overload or fail to deliver a measurable response, so that actually the receiver excitation is adjusted in 20 db steps to some convenient intermediate level, and the output signal value is then weighted by the input variations to provide the equivalent of a constant input response.

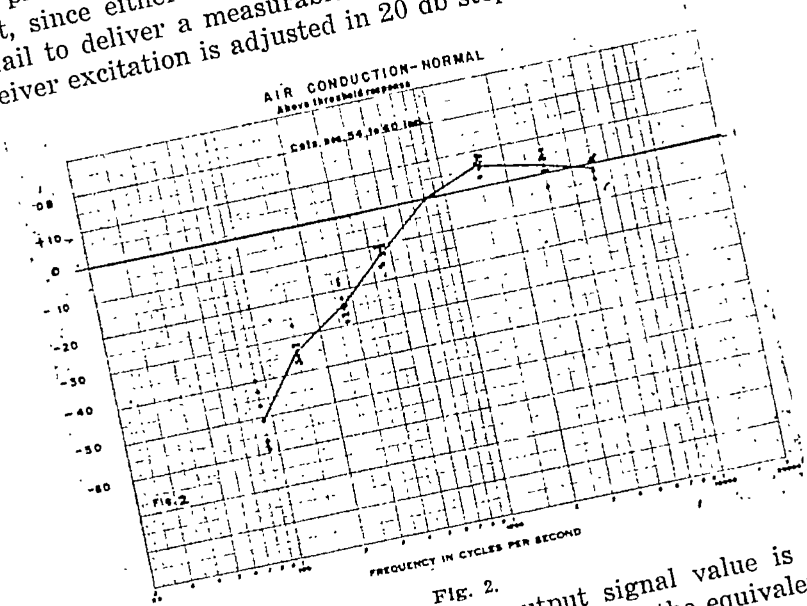

Fig. 2.

TYPICAL RESPONSE CHARACTERISTICS OF THE CAT.

Fig. 2 shows the measured normal air conduction response of seven cats at frequencies from 90 to 5,000 cycles per second, which covers that portion of the audible spectrum most important to speech intelligibility. A line has been drawn through the average of these seven test values, which are made to agree at 800 cycles, and it will be apparent that the deviation of the individual points from the mean is not greater than should be expected. This average characteristic is, of course, a summation of the animal's response and the receiver characteristic, but if the same receiver is used on succeeding tests, then any difference appearing will be due to changes in the acoustic response of the animal.

Fig. 3 is an identical set of tests using the bone conduction receiver. It will be noted that the spread of individual test points about the mean characteristic is much greater than with air conduction. This is due to the difficulty of duplicating the exact pressure and angle of incidence between the skull and the bone conduction unit in different animals. The receiver is applied directly to the temporo-squamosal area, and is sewn in place in order to reduce the variables as much as possible.

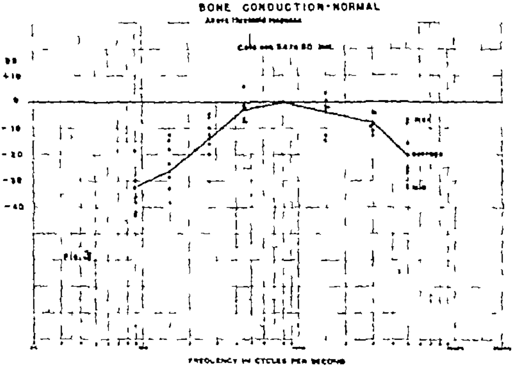

FIG 3

In Fig. 1 the average normal air conduction characteristic of Fig. 2 has been plotted as a straight line at zero db in the manner customary in human audiometry graphs. Thus, departures from the normal are more readily apparent, both as to kind and degree. Curve "A" of Fig. 4 shows an average of the changes occurring when the round window is blocked with dental cement (zinc phosphate cement powder). The average difference from normal proves to be a nearly straight line with maximum attenuation at the low frequency end. This is possibly due to the fact that the amplitude of the stapes excursion (and thus of the perilymph) is inversely proportional to the frequency when the applied acoustic energy is constant; therefore, the motion of the round window membrane would tend to be greatest at the low frequencies, and the loss from blocking should thus be greatest at this end of the spectrum.

Curve "B" of Fig. 4 indicates the departure from normal when the oval window is blocked. and shows not only much greater attenuation than does Curve "A," but also reverses the change of attenuation with frequency. In this case we are blocking the point of entry to the cochlea, and thus the various elements which couple this point to the diaphragm of the receiver are working into a very stiff load. There are a number of compliances in or between these elements: the air column between the receiver and the ear drum, the coupling between malleus and incus, the axis about which the malleus and incus rotate, and the coupling between incus and stapes. The acoustic energy will be partially dissipated by the yielding of these compliances before reaching the perilymph. This constitutes a shunt for the acoustic vibrations, and the low frequencies of relatively high amplitude are shunted less by such compliance than are the high frequencies.

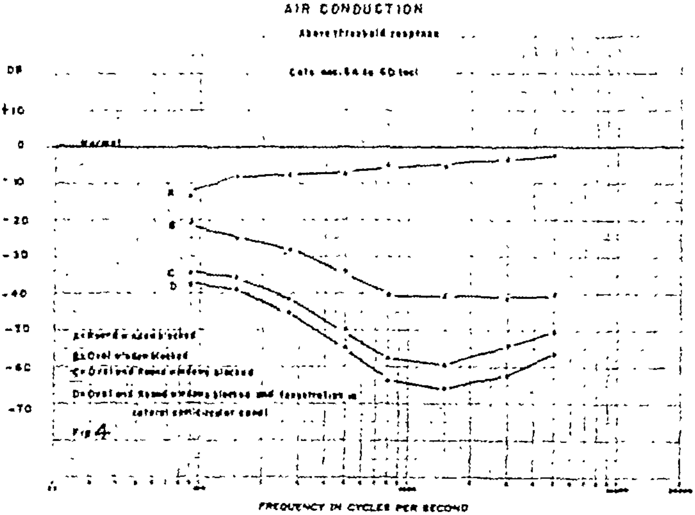

Fig. 4.

Blocking both the round and oval windows should apparently produce a response characteristic which is the algebraic sum of curves "A" and "B"; however, we find curve "C" develops considerably more than the expected attenuation, particularly in the mid-frequencies. There appears to be no ready explanation for this response characteristic unless some, or all, of the curves are partially in error. Since the number of samples is relatively small, some shift in amplitude or form will likely occur as additional data is acquired.

Curve "D" roughly parallels curve "C" and is the response characteristic obtained after fenestrating the lateral semicircular canal, with both round and oval windows still blocked. This is a condition which has no clinical parallel, since in curve "D" we are still applying the acoustic energy to the stapes as in curve "C." The "fenestra" then acts as a partial by-pass for what little energy passes into the perilymph through the blocked stapes, and the additional attenuation of curve "D" results from this by-pass action.

In considering the curves "B," "C," and "D" of Fig. 4, it may be worth noting that these curves as measured show a fairly linear rising characteristic, but since they do not rise as fast as the normal characteristic of Fig. 2, they appear in Fig. 4 to fall from low to middle frequencies.

Bone conduction tests were made at the same time as the preceding air conduction tests, and are shown in Fig. 5 plotted in the same manner as the air conduction characteristics of Fig. 4.

The mechanics of bone conduction have not been very definitely established, but the generally accepted theory is that vibrations applied to certain areas of the skull cause minute deformations of the bony labyrinth, which in turn cause movement of the perilymph and resultant excitation of the basilar membrane. Assuming this theory to be correct, the vibratory energy can not be said to enter the cochlea at any specific point nor travel through any fixed path within the labyrinth, and it is thus exceedingly difficult to rationalize the role of the various inertias, compliances and frictions which determine the response of the cochlea in bone conduction. What appears to be significant, in these measurements, is not the frequency response, but the variation in bone conduction level in the mid-frequency range with the various degrees of window blocking, while the neural sensitivity is known to be the same in all cases.

K. R. Smith[16] found essentially the same mid-frequency loss in bone conduction when the stapes was immobilized by means of a thread attached to the crura. The rigidity of fixation which we obtained by the use of dental cement is, however, somewhat greater as evidenced by increased loss, particularly in the 2000 and 3000 cycle region.

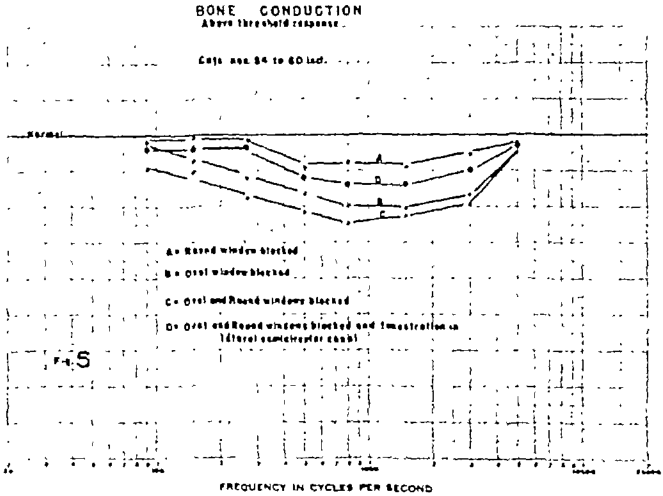

Fig 5.

Bone conduction is used as a measure of auditory nerve response, particularly in predicting the probable outcome of fenestration; yet in the mid-frequency range where the important speech frequencies are found, the bone conduction as we measure it, and thus the apparent neural response may vary within a 25 db range, depending on the degree and kind of window mobility. This factor has been recognized indirectly by Carhart[12] and others in noting that bone conduction in the mid-frequency range usually improves after fenestration. Curve "D" shows this improvement, although not in exactly the same form as the "Carhart notch."

Another factor brought out by these tests, is more or less the inverse of the bone conduction characteristics just noted, i.e., that the most severe window occlusion which we obtained can contribute only about 25 db attenuation from normal in the mid-frequencies; therefore, losses greater than these in bone conduction response or loss at the high frequency end should, probably, be attributed to neural degeneration.

All of the above has been based upon two assumptions: *1.* That the character of the data presented will not materially change as the number of samples is increased; *2.* That the auditory responses of the cat are essentially the same as the human response in the frequency range measured.

CONCLUSIONS.

It is not intended to draw far-reaching conclusions from this preliminary experimental material. We are not completely justified in equating cat and human cochlear responses; however, the following considerations may be of value:

1. Supra threshold frequency analyses of cochlear microphonics in experimental labyrinthine deformations may be helpful in cochlear physiologic studies.

2. Bone conduction studies under these circumstances may yield valuable information.

3. Variables simulating changes in clinical otosclerosis may be studied by this technique.

4. Labyrinthine occlusions may account for bone conduction deficits of 25 db without concomitant change in cochlear neural function.

Further studies in this and related areas are underway.

SUMMARY.

Quantitative studies of supra-threshold cochlear microphonics in experimental labyrinthine deformations are being carried out with bone and air conducted sound. Bone conduction deficits of 25 db may be attributed entirely to labyrin-

thine occlusions. It is hoped that some of these studies will have clinical significance in the problem of fenestration surgery.

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RYL IN THE CONTROL OF VERTIGO.

Preliminary Report on Its Clinical Use.*

DAVID D. DEWEESE, M.D.,
Portland, Ore.

past four years there have been increasing experi- and clinical observations regarding the effectiveness tertiary amines—dramamine, benadryl hydrochloride thorazine, in the control of nausea and vomiting. There some evidence that banthine, which is a quaternary amine, may have a similar action. The high percentage of reported good results obtained with these drugs in the control of vomiting has led to their use in the control of vertigo.

There are a number of reasons for the effectivness of these drugs. Thorazine and benadryl depress the chemoreceptor trigger zone in the floor of the fourth ventricle known as the vagal trigone. They are effective in blocking vomiting caused by apomorphine, morphine, and ergot.[1] They do not depress vomiting caused by local gastric irritation or by substances which cause vomiting by action on the ganglion nodosum or the frontal lobe. Both these drugs prevent motion sickness; therefore, they must have some effect on the central network which is involved in labyrinthine reaction.[2]

After benadryl has been administered a larger amount of electrical current is required to stimulate the medullary vomiting center (dorsolateral portion of the reticular formation and the tractus solitarius).[3]

Physiologically, the complicated system involving the labyrinth, the vomiting center and the vagus nerve is known as a cholinergic system. Experimental stimulation of this system produces unilateral miosis, compulsive turning, horizontal nystagmus and emesis. These reactions are known as the "adversive syndrome." They can be altered and diminished by

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* Read at the meeting of the Western Section of the American Laryngological, Rhinological and Otological Society, Inc., Portland, Ore., February 6, 1954.

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BENADRYL IN THE CONTROL OF VERTIGO.

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DAVID D. DEWEESE, M.D.,
Portland, Ore.

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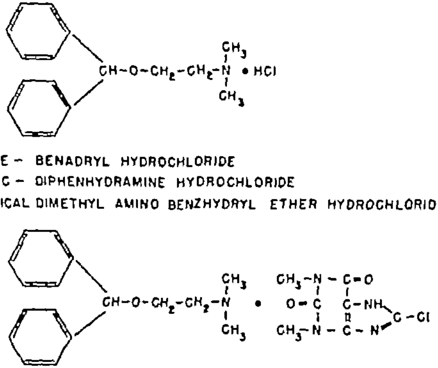
My use of the drug has been restricted to 50 and 100 mg doses, given intravenously This has been administered either directly in 10 cc of sterile saline, or water, or by adding 100 mg to an intravenous solution of saline or glucose I have administered the drug in two ways *1* injection of the contents of a 10 cc ampule slowly, that is, over a period of one to three minutes, 2 addition of a 10 cc ampule to an intravenous infusion of 500 to 1,000 cc. of saline or glucose The latter method is obviously much more convenient, and appears to be effective



TRADE - BENADRYL HYDROCHLORIDE
PUBLIC - DIPHENHYDRAMINE HYDROCHLORIDE
CHEMICAL DIMETHYL AMINO BENZHYDRYL ETHER HYDROCHLORIDE

TRADE - DRAMAMINE
PUBLIC - DIMENHYDRINATE
CHEMICAL - 8-CHLOROTHEOPHYLLINE SALT OF DIMETHYLAMINO BENZHYDORYL ETHER

Fig 1—Part 1.

Because benadryl is highly soluble it is rapidly absorbed and should be effective by mouth in patients who can tolerate a large enough dosage by this route, however, nausea and vomiting preclude its use by mouth until, or unless, the nausea or vomiting is first controlled by intravenous administration of the drug

As all of us who do fenestration surgery know, that in atropine. It has been shown that thorazine, dramamine, and benadryl exert a similar action; therefore, it is concluded that these drugs have a central atropine-like action rather than an effect on the peripheral cholinergic fibers to the stomach.[4]

Although there is one report[5] which maintains that dramamine may prolong the latent period and increase the duration of nystagmus following caloric or galvanic labyrinthine stimulation, these findings have not been substantiated. Winston, *et al.*,[6] could not find any significant alteration of nystagmus by either dramamine, benadryl or thorazine; however, all three drugs blocked nausea and vomiting associated with nystagmus, and appeared to provide subjective relief of vertigo.

In 1951 McCawley, *et al.*,[7] at the University of Oregon Medical School, injected benadryl intravenously in various dosages for control of nausea and vomiting, which, *1.* followed various operative procedures, and which *2.* occurred in certain medical conditions. One series of patients were given benadryl prior to operation; another received benadryl when vomiting began. The most effective dose without undue side effects was 100 mg., which amount is available in a 10 cc. ampule of distilled water.

Of the patients in the control series, 50 per cent had one or more episodes of vomiting and a high incidence of nausea; but only one out of more than 70 patients who received the benadryl had any vomiting. Most of the patients in the latter group were free from nausea during the postoperative period.

As a result of the investigations of McCawley, *et al.*, I decided to attempt the use of benadryl intravenously in the control of vertigo, regardless of its cause. It was not a controlled investigation. During the past two years I have given the drug to patients with the following conditions:

1. Vertigo following fenestration surgery.

2. Nausea and vomiting after temporal bone surgery.

3. Acute vertigo from labyrinthine hydrops.

4. Vertigo due to suppurative labyrinthitis.

of glucose. The other four received the benadryl in 10 cc. of water administered over about three minutes.

Eight patients, including the one who received only 50 mg. of the drug, had no further vomiting, and five were relieved of nausea. The remaining three had only slightly residual nausea.

Two patients had recurrent nausea and vomiting, 12 and 24 hours, respectively, after the benadryl had been given. Both these patients received a second dose of 100 mg. benadryl intravenously in 500 cc. of 5 per cent glucose. Following the second dose there was no further nausea or vomiting.

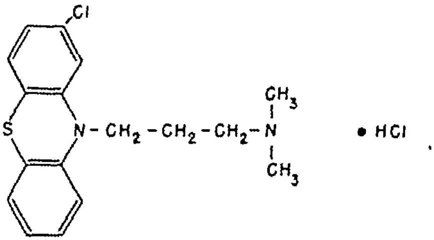
All 10 patients received 50 mg. dramamine by mouth every six hours during their hospital stay. Dramamine in this dosage is given routinely postoperatively to all my patients having fenestration surgery.

Four of 26 patients who had radical mastoidectomies exhibited enough nausea and vomiting postoperatively to require treatment. Each had a labyrinthine fistula caused by cholesteatoma. The nausea and vomiting of two patients was promptly controlled, without recurrence, after administration of 100 mg. benadryl intravenously. The other two had relief from nausea and vomiting but remained unsteady and subjectively dizzy.

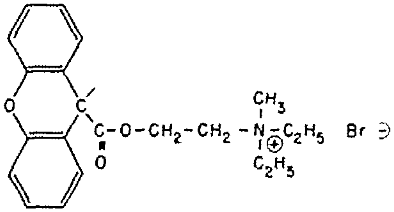
In the acute attack of labyrinthine hydrops nausea, vomiting and subjective dizziness should be controlled, if possible. This is desirable because it assures the patient that his symptoms can be controlled. Although we all know these attacks are self-limited, and that they can be stopped at times by adrenalin, atropine, or histamine, the use of benadryl may offer a safer and more convenient means of control.

Under my direction two such patients were given 100 mg. benadryl intravenously during acute attacks: one received the drug on two occasions, the other on one occasion. The results were most encouraging, since the attack in each instance was terminated within thirty minutes.

spite of improvements in technique and routine administration of chemotherapy postoperatively, there still are a fair number of patients who have enough postoperative dizziness and vomiting to delay their ambulation. Many of these patients have a history of motion sickness. Dramamine given orally is effective in controlling the nausea and dizziness of most of these patients, but it cannot be used if there is excessive nausea and vomiting.



TRADE - THORAZINE
PUBLIC - CHLORPROMAZINE
CHEMICAL-10-(γ-DIMETHYLAMINO PROPYL)-2-CHLORO PHENOTHIAZINE HYDROCHLORIDE



TRADE - BANTHĪNE BROMIDE
PUBLIC - METHANTHELNINE
CHEMICAL- DIETHYLAMINOETHYL XANTHENE-9-CARBOXYLATE METHOBROMIDE

Fig. 1.—Part 2.

In the past two years, 10 of 51 patients on whom I did fenestration operations had such symptoms. They exhibited six to eight attacks of emesis on the day of operation, or continued to have as many as 12 to 14 attacks of emesis the morning after operation. Nine of the patients were each given 100 mg., and one was given 50 mg., benadryl intravenously from six to 36 hours after operation. Of these, six were given the drug in an intravenous infusion of 500 to 1,000 cc.

any doubt in the minds of these respective patients, that they felt less dizzy, that they were able to sit up, and in many cases, that they were able to walk after their nausea and vomiting had been relieved.

I do not know whether this implies that stimulation of the medullary vomiting centers produces a subjective feeling of dizziness. It reminds me of the popular quotation during the late 1920's of "which comes first, the chicken or the egg?" There can be little doubt that benadryl administered in the manner and dosage described. is well worth using in the conditions mentioned, and perhaps, in other allied conditions.

Medical control of the symptoms arising from disturbance of the parasympathetic nervous system is a goal which has been long sought, and dizziness is frequently such a symptom.

Although this discussion is concerned with benadryl alone, further work should be done with dramamine, thorazine, banthine, hydergine ®* and other pharmacologically allied drugs, for the control of dizziness. The purpose of this preliminary report is to attempt to stimulate such studies. Results of well controlled experiments in large numbers of patients may make possible future treatment of dizziness on a sounder basis.

SUMMARY AND CONCLUSIONS.

Benadryl was tried for control of nausea and vomiting and associated dizziness in 14 patients subjected to temporal bone surgery, two patients with labyrinthine hydrops, and one patient with suppurative labyrinthitis.

From this preliminary study it is concluded that:

1. Benadryl given intravenously is effective in controlling nausea and vomiting and subjective dizziness.
2. Further studies with this and allied drugs should be carried out.

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One patient with acute suppurative labyrinthitis was treated in the same manner. I first saw him in April, 1951. He gave a history of having had five mastoidectomies on his right ear, and stated further that for two weeks prior to admission he had persistent pain in this ear.

Examination showed chronic suppurative otitis media in the right ear, with bloody purulent discharge. Culture revealed the infecting organisms to be streptococcus hemolyticus and staphylococcus aureus.

On April 5, 1951, a radical mastoidectomy was done. The postoperative course was uneventful. Following his discharge convalescence continued well until April 21. At this time he had serosanguineous discharge from the ear that had been operated on, as well as loud tinnitus in the ear. There was also rapid onset of severe whirling vertigo, with nausea and vomiting. He could not stand without help, and tended to fall to the left and backward. He had gross rotary nystagmus to the left; however, he was oriented. There were no localizing neurologic signs.

He was hospitalized immediately and was given intramuscular injections of penicillin and intravenous injections of sulfadiazine. Over the next five days he also received benadryl intravenously in doses of 50 mg. approximately 12 hours apart. After each administration in this dosage the nausea and vomiting were controlled for from six to 14 hours. Even during the active nausea and vomiting of the first five days, he could be relieved enough to sit up and take food by mouth. Without the benadryl this was impossible, because he had violent nausea and vomiting which persisted until the drug was again administered. After the fifth day benadryl by mouth controlled his nausea and vomiting, and appeared to benefit his subjective dizziness, but if he went without the drug for part or all day he always seemed to feel unsteady.

Drowsiness was produced in some patients. In a few there was a subjective feeling of warmth and flushing of the skin, but both these effects were of short duration. In none of the 17 patients was there any serious side effect.

DISCUSSION.

In reporting the results of the effect of benadryl given intravenously to these 17 patients, it is obvious that little has been said regarding the control of vertigo, which is the title of this paper. The results presented confirm the already known control of nausea and vomiting by this drug. This immediately raises a question as to how much influence the nausea and vomiting have on a subjective feeling of dizziness and, therefore, to what extent dizziness is relieved by benadryl.

As I have already stated, this was not a controlled experiment. I did not attempt to measure nystagmus, either during or immediately following the administration of benadryl. Nevertheless, there is no doubt in my mind, nor was there any doubt in the minds of these respective patients, that they felt less dizzy, that they were able to sit up, and in many cases, that they were able to walk after their nausea and vomiting had been relieved.

I do not know whether this implies that stimulation of the medullary vomiting centers produces a subjective feeling of dizziness. It reminds me of the popular quotation during the late 1920's of "which comes first, the chicken or the egg?" There can be little doubt that benadryl administered in the manner and dosage described, is well worth using in the conditions mentioned, and perhaps, in other allied conditions.

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The Alexander Graham Bell Association for the Deaf (formerly The Volta Speech Association for the Deaf) will hold its 1954 Summer meeting in St. Louis, June 14-18 inclusive. The theme of this meeting is "Let's Face the Issues," and some of the questions to be discussed are: "Where should the deaf child receive his education?" "What are the possibilities and limitations of auditory training?" "What are the principal issues in teaching speech?" and "What contributions can be made to the understanding of the deaf child by persons in peripheral fields?"

All sessions will be held at the Chase Hotel, but there will be Open House at Central Institute for the Deaf and St. Joseph Institute for the Deaf.

For further information, address: Robert Goldstein, 818 S. Kingshighway, St. Louis 10, Missouri.

GENERAL ANESTHESIA IN OTORHINOLARYNGOLOGY *†

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Many operative procedures done by members of your specialty are completed with local anesthesia, either local infiltration, sometimes nerve block, or topical application Local anesthesia, when used correctly in safe amounts, has probably less systemic effect than other anesthetics When a given operation can be accomplished with a safe dose of local anesthetic agent, without undue discomfort to the patient and with ease by the surgeon, local should be given consideration as first choice

Topical anesthesia is, of course, not without its disadvantages and dangers. We all know of even fatal reactions to topical anesthesia, so that development of a topical agent of lesser toxicity would be of great value When it was shown that one of the pharmacological properties of antihistaminic drugs was the ability to produce local anesthesia, it was hoped that here might be found an agent capable of producing safe and adequate anesthesia, however, some investigation is discouraging In a report by Mathieu & Crandall,[1] on the use of 4 per cent pyribenzamine in only 12 cases of bronchoscopy, several disadvantages were listed

1 In only two cases was anesthesia completely satisfactory, using pyribenzamine alone

2 Salivation was increased

3 An unpleasant burning sensation in the mouth and neck was experienced by all patients

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† From Anesthesia Department Emanuel Hospital Portland Ore

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4. In two cases, untoward reactions resembling those following the use of cocaine occurred.

Perhaps further investigation will lead to something more encouraging.

CHOICE.

A question frequently asked concerning general anesthesia goes something like this: "Mr. Smith has so-and-so. What shall we give him?" Or: "Don't you think she is all right for a little pentothal?" To make a choice of anesthesia we should consider the history of the patient, his physical and mental status, the laboratory work, the site and extent of the operation, the desires of the surgeon, and the abilities of the anesthetist. In general, we can say that the importance rests more upon the administration than upon the drugs selected.

AGENTS.

We do have a number of anesthetic agents available, each with its own peculiarities. These characteristics may be of minor importance in some cases but of marked importance, even to contra-indication of use, in others. To mention a few commonly used agents: nitrous oxide is a non-toxic gas which has no definite contra-indications to its use; however, it is a weak anesthetic agent, and the harmful effects of hypoxia may be produced in attempting to provide complete anesthesia with nitrous oxide alone, rather than by supplementing it with some other agent.

Cyclopropane provides complete anesthesia with a generous oxygen concentration, but it is explosive and should not be used with the electro-cautery or other electrical equipment. It must not be used with epinephrine or its related drugs, which is an important consideration in your field. Cyclopropane not uncommonly produces arrhythmias. Epinephrine further enhances the irritability of the cardiac automatic tissue, and the use of the two together may result in ventricular fibrillation.

Ethyl-ether is used much less frequently than it was 10 or 15 years ago, but it probably still has the greatest margin of safety. Some cardiac irregularities may occur during ether anesthesia, but they are not of serious nature. Ether may well be chosen in the presence of fibrillation, heart block or coronary disease. Ether is a sympathomimetic agent producing bronchial relaxation, so that its use is indicated with asthmatics. On the negative side, it produces some metabolic changes, more than other agents. Liver and kidney function may be decreased, acidoses may appear, nausea and emesis are common, and most patients do not like it.

Thiopental and Surital are agents most commonly used for intravenous anesthesia. They are rapid acting hypnotics which depress the central nervous system. They also depress respiration and the vasomotor center. They seem to enhance the laryngeal reflexes and laryngospasm occurs not infrequently, but they produce no definite organ damage and, when used in moderate amounts, combined with oxygen and nitrous oxide, and perhaps a muscular relaxant, find rather wide use, especially when a non-explosive anesthetic is needed. They provide a very pleasant inducing agent, which patients love and frequently ask for. Other agents have similar individual properties, which are to be considered in the choice of an agent.

METHODS.

The choice of agent for ear, nose and throat surgery may be the same for a given patient as for other types of surgery, but the method of administration requires special consideration. Since the surgery is done about the head and neck, we must make the anesthesia safe for the patient by providing a continuous free and open airway, and must prevent aspiration of blood or foreign materials. We must also provide adequate operative area for the surgeon and not have any of our equipment in the way. These requisites are best provided by an endotracheal technique, adapted to the particular operation to be performed.

For nasal operations oral intubation may be done, and the tubing to the gas machine led down over the chest to the side of the table. The anesthesiologist then takes his position at the side of the patient, perhaps nearer the foot than the head.

The patient's color and pulse are watched in the hands or feet. The character of respirations are followed by watching the breathing bag. Blood pressure readings are recorded at regular intervals. Intravenous fluids may be started in an ankle vein. Even though we are removed from the normal position of the anesthetist, we may carry on a safe, satisfactory anesthesia, out of the way of the surgeon.

For operations in the oral cavity, nasal intubation may be done and the tubing brought up over the forehead of the patient, leaving the mouth free and still obviating the danger of respiratory obstruction or aspiration.

Tonsillectomy and adenoidectomy are the most common operations done in the oral cavity. The majority of adult tonsillectomies are done under local anesthesia. Those done under general anesthesia are usually intubated. Tonsillectomy and adenoidectomy in children are frequently done using insufflation anesthesia. They may also well be done with an endotracheal technique, especially those operations of longer duration. Some surgeons take 10 to 15 minutes, while others may take an hour. Against the use of the endotracheal tube is the fact that those frequently administering tonsil anesthesia are not adept at intubation and also that some surgeons complain of the tube being in the way. Barton,[2] of California, has recently described an endotracheal tongue blade adaptable to the Crowe-Davis or the McIvor mouth gag, which is said to afford better exposure. A metal tube is incorporated in the tongue blade, and the endotracheal tube is threaded through it and held down in the midline against the tongue.

Peroral endoscopy is usually a short procedure, but one which may test the skill of the anesthetist. Bronchoscopy in children may most safely be carried out with ether inhalation and insufflation. This type of anesthesia may also be used in adults, but induction time is slow and may take longer than the endoscopy. If the procedure is of some length it may be difficult to maintain adequate anesthesia, or if the patient is deeply anesthetized, recovery time is prolonged with slow return of pharyngeal and cough reflexes. Other methods also have disadvantages.

One of our newer muscular relaxants, succinylcholine, is finding use in this field.[3] Succinylcholine produces relaxation of rapid onset and of very short duration. One method of using this agent may be described: topical anesthesia is first applied to the pharynx and larynx; then, when the bronchoscope and equipment are in readiness, the patient is anesthetized with intravenous thiopental or surital and a 0.1 per cent succinylcholine drip started. Oxygen may well be given during induction. In five minutes or less, the surgeon may proceed. Succinylcholine here is superior to curare preparations because its action ceases soon after administration is stopped, and because the cords will be found to be more in abduction; however, it is not a perfect method and must be treated with marked respect. The more pentothal given, the more will be the resulting respiratory depression, and an excess of succinylcholine will produce apnea.

For operations such as laryngectomy, the initial surgery may be accomplished with an oral tube in place. When the trachea is transected an ordinary cuffed tube or an angled modification,[4] which may give the surgeon more room, is inserted into the new tracheal opening and the anesthetic continued to the end of the operation without fear of obstruction or aspiration of blood.

For some operations on the larynx, it may be an extreme advantage or a necessity to perform a tracheotomy first. During the past ten years, tracheotomy operations have been performed with increasing frequency. As has been pointed out, by Davis, *et al.*,[5] tracheotomies are now recommended as a prophylactic measure and are no longer regarded as a last resort for dire emergencies. The safety of the patient is our first consideration and, if a tracheotomy is going to make the operation or anesthesia safer for the patient, it should certainly be done. In such as a Kelly operation, a tracheotomy may be done under local anesthesia, or under general anesthesia over an endotrachial tube. When the trachea is reached and the cartilages are to be cut, the oral tube is removed and a modified tube inserted into the stoma. The operation may then be peacefully continued, with the assurance that we have the air-way under perfect control. We have made the patient safer for the operation.

POSTANESTHESIA RECOVERY ROOM.

Efforts to increase the safety of the patient must not cease with the completion of the operation. The immediate postoperative period following general anesthesia may be one of the most critical times of the patient's hospitalization. He must be watched closely until there is a return of consciousness and of the protective reflexes, if early postanesthetic complications are to be avoided. We attempt to have the patient as much awake as possible at the termination of the surgery, so that this period is not a prolonged one; even then, it is difficult to obtain adequate care when the patient is returned to the surgery floors.

The answer to this problem is the use of a postanesthetic observation room.[6,7,8] To this space surgical cases may be returned for close observation by trained personnel until it is felt safe to return them to their beds. This room should, preferably, adjoin surgery, so that the anesthesiologist or surgeon may be readily available, and may slip in to check on the patient between cases. Ready for immediate use are oxygen, suction, drugs, fluids, and other supplies which may be needed. It is most helpful in our care of the unconscious patient, and provides continuity to the program of ever watching for aids to increase the safety of the patient. If you do not have a postanesthetic room in your hospital, I would recommend it to you.

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THE BRONCHOSCOPIST'S AID TO THE THORACIC SURGEON.*†

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Because of the remarkable advances in chemotherapy during the last fifteen years, the character of otolaryngologic practice has changed. Fulminating infections are exceedingly rare now, and the young otolaryngologist is fortunate if during his training he sees one patient with meningitis, lateral sinus thrombosis or osteomyelitis of the frontal bone or superior maxilla. We otolaryngologists, therefore, are directing our attention to preventive medicine, early diagnosis of disease and corrective surgical procedures.

One of the major medical problems confronting every physician at the present time is that of carcinoma. The otolaryngologist has assumed a prominent place among surgeons treating cancer and is confronted with this condition more and more, not only in the performance of surgical procedures in the region of the head and neck, but also in the assistance he gives to the thoracic surgeon. The otolaryngologist's knowledge of the respiratory tract can be of considerable value in providing the thoracic surgeon with information he needs for successful excision of the neoplastic tissue. He can be of particular help in the bronchoscopic study of the patient. He is far better qualified than others to serve as the bronchoscopist on the chest service; and along with the radiologist, internist, pathologist, and thoracic surgeon, he fulfills an important place in a team which is required for the best care of patients with diseases of the chest. It behooves us then, to increase our knowledge and improve our skill of bronchoscopy, so that we will remain an indispensable part of this working team in the treatment of thoracic diseases.

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The great increase in the number of reported cases of carcinoma of the lung in recent years has caused much speculation as to whether the incidence of the disease has actually increased or whether more cases are being recognized because of better diagnostic studies. Approximately 10 per cent of all cases of primary carcinoma originate in the lung, which ranks second only to the gastrointestinal tract as the most frequent site for cancer.

In spite of the progress made in the methods of recognition of cancer, as well as the fact that not only the medical profession, but also the layman are becoming more "lung cancer conscious," we are still seeing a large number of patients whose disease is so far advanced as to be inoperable. It has been stated by some that the majority of carcinomas of the lung originate in the main or branch bronchi, and usually by the time the patient is sufficiently ill to seek medical advice, operative removal is impossible. Others, however, believe that tumors of the main bronchi give rise to symptoms early and spread slowly, so that the patient consults the surgeon in time for surgical removal. These diametrically opposed view-points are both true, because there are many patients who consult a physician in the early stages of the disease but the physician neglects to carry out a thorough examination. Too often the physician is inclined to disregard the early symptoms and wait for more definite physical or roentgenologic evidence to develop. By this time the disease has usually progressed beyond surgical aid.

The most persistent symptom of bronchogenic carcinoma is chronic cough, which at times may become blood tinged. Unfortunately, the importance of this symptom is too often minimized, both by the patient and by the physician. The fact that bronchogenic carcinoma can masquerade under a chronic cough, which at times may become blood tinged and is productive of some mucous should be kept in mind at all times, and in these patients a thorough investigation is mandatory if bronchogenic carcinoma is to be diagnosed at a time when surgical removal is possible. Our clinical pace must be quickened.

Another factor responsible for delay in diagnosis is the fact that there are no characteristic symptoms of bronchogenic carcinoma. The commonest symptom is a chronic persistent cough, productive of mucous, and at times slightly blood tinged. It appears early before the bronchi have become occluded from irritation and partial obstruction of the air passages. At this time physical examination usually reveals no abnormalities, and roentgenologic evidence is either absent or inconclusive; however, the diagnosis can usually be made at this time by bronchoscopic examination. If only a small part of the lung and bronchial tree is involved, the diagnosis can be made only if the bronchoscopist has a thorough knowledge of the finer anatomic relationships of the lung. The openings of the segmental bronchi can be seen and identified through the bronchoscope.

Later, as the cough becomes more persistent, instead of blood tinged sputum, hemoptysis may develop. This sign is a definite indication for bronchoscopy, even if the patient has tuberculosis, and especially in those patients in whom no tubercles have been demonstrated.

Dyspnea usually develops after the bronchus has become obstructed and may at times be quite severe, even though only a small portion of the lung is involved. Cardiac disease must be considered in the differential diagnosis; also, a diagnosis of asthma must not be made without investigating the possibility of carcinoma, because some wheezing is usually associated with dyspnea caused by an obstructing tumor.

Pain does not develop until the advanced stages of bronchogenic carcinoma, except in those patients with pleural involvement whose disease originates in the periphery. Similarly, fever is a late manifestation, but may appear early if obstruction and infection develop distal to the growth. Emphysema, obstructive atelectasis and pulmonary abscess appear in the terminal phase of the disease, and can be demonstrated on physical and roentgenologic examination at that time.

The value of bronchoscopy in the early diagnosis of bronchogenic carcinoma cannot be too strongly emphasized. It is almost unanimously agreed that this is the one procedure which makes possible early recognition of the condition. In most cases the diagnosis can be made earlier and more positively by bronchoscopy than by any other available means. This, of course, does not mean that this should be the only means of diagnosis. These patients should be studied carefully by the internist, radiologist and thoracic physician, as well as the bronchoscopist.

It is advisable that bronchoscopy be performed under local anesthesia in order to obtain the cooperation of the patient during examination of the segmental bronchi and to control hemorrhage should it result from biopsy or manipulation of the growth. All of our bronchoscopies were performed under local anesthesia with no bad results and with no complications which would delay operation. The patient is given some form of barbiturate the evening before, and again one hour before bronchoscopy is to be performed. A 10 per cent solution of cocaine is sprayed into the mouth, and the patient is instructed to expectorate the solution after it has been held in the mouth for a few seconds. He is especially cautioned not to swallow any of the spray. A laryngeal cotton applicator is then dipped in a 10 per cent solution of cocaine, the excess solution being squeezed out. The applicator is applied to each pyriform fossa and kept there for exactly five minutes. A cotton pledget saturated in this cocaine solution and thoroughly wrung out is placed between the upper gum and lip and also kept there for five minutes. The patient is then made to sit up and 1 cc. of a 1 per cent solution of pontocaine is instilled into the larynx, with the patient tilting slightly to the right; this procedure is then carried out with the patient tilted to the left. He is then placed on the regular operating table and draped, after which bronchoscopy is carried out without a headholder. Usually the 7:40 or 7:35 Jackson bronchoscope may be inserted directly into the larynx. Only occasionally does one have to use the laryngoscope as an aid.

Once the bronchoscope has entered the trachea the examination begins, because in addition to obtaining a biopsy specimen the bronchoscopist can obtain information which will enable him to give the thoracic surgeon a full description of the tracheobronchial tree This will be of considerable help to the thoracic surgeon when he removes the growth

The trachea is inspected for shift or displacement and for fixation here or at the carina The bronchoscope is then directed into the right bronchus, which is examined for size, compression or fixation The opening to the lobes and the segmental bronchi are then inspected If a growth is found in a definite lobe of bronchopulmonary segment, it is definitely identified and the distance to the main bronchus measured If the main stem bronchus is involved, then the distance to the carina should be measured in order to determine whether there is enough clearance for removal of the lung Although a definite diagnosis cannot be made in many instances, much valuable information can be given to the surgeon, which, along with the physical and roentgenologic findings make exploratory thoracotomy possible Thus, it might be possible for the surgeon to remove a malignant tumor which might be inoperable by the time a positive biopsy is obtainable

Besides biopsy and aspiration for examination for neoplastic cells, the bronchoscopist can supply additional valuable information which can be obtained by no other means Spreading of the carina, and distortion and fixation of the bronchi are important indications of involvement. Absence of expansion and elongation on inspiration and contraction on expiration indicates peribronchial involvement A rigid tracheobronchial tree also denotes peribronchial involvement Frequently, obstruction to the main or secondary bronchus, due to proliferation of the growth, can be removed sufficiently to establish drainage or an adequate airway which results in improvement in the general condition of the patient prior to operation This may necessitate postponing operation because several bronchoscopic procedures may be required, but the short delay is offset by the improvement in the general condition of the patient. It may also make possible collapse of the lung at the time of operation and thus provide much better surgical exposure Even if a biopsy specimen cannot be obtained in these patients, often bloody or purulent drainage from one of the segmental bronchi is seen, and this localizes the lesion to a definite area; moreover, the secretion may be aspirated for study of malignant cells. If no definite pathologic diagnosis can be made following bronchoscopy and other examinations, then from the bronchscopic observations, roentgenographic findings, and the history and physical findings, the thoracic surgeon may consider exploratory thoracotomy indicated, which in many cases at this early stage means successful removal of a malignant growth.

Carcinoma of the lung is being recognized more often, not only because of the more adequate diagnostic facilities but also because of the establishment of cooperation between the internist, roentgenologist, pathologist, bronchoscopist and thoracic surgeon. The bronchoscopist plays a vital role in the early diagnosis of this condition. It, therefore, is our obligation *to become proficient bronchoscopists so that we can* supply the thoracic surgeon with all the information he needs for successful excision of these malignant growths.

Although the early manifestations of pulmonary carcinoma are indefinite, a chronic, persistent cough, often productive of mucous which at times is slightly blood tinged, is often the first indication of trouble, just as persistent hoarseness is the warning signal of carcinoma of the larynx. Bronchoscopy is, therefore, mandatory in all patients with a persistent chronic productive cough if bronchogenic carcinoma is to be diagnosed at a time when it is still operable.

CONSERVATION OF HEARING IN THE MODIFIED RADICAL MASTOIDECTOMY.*†

Technical Considerations.

SHIRLEY HAROLD BARON, M.D.,
San Francisco, Calif.

It is generally agreed that the main objectives in mastoid surgery are to eliminate dangerous pathology and to preserve function, providing that saving function can be done at no expense to the patient's safety.

Just what constitutes safety? To this question there will be no uniform reply, but if we try to answer it without prejudice we must unshackle ourselves from the precept of times gone by. We are suffering from a "hang-over" of another era, when complications were rampant, when antimicrobial medicaments were nonexistent, when techniques were less advanced, and when radical mastoid surgery was the rule and the less radical, the exception.

There still persists a tendency toward radical surgery, very likely because of our continued obsession with the intracranial complications, which as Tumarkin[1] aptly stated, "loom up too largely in our minds and in our textbooks. Whatever may have been the case in the past, today they are interesting rareties."

When we shall rid ourselves of this obsession and the overwhelming compulsion "to take out a little more," when doing a little less would suffice, conservative mastoid surgery, with a good functional result, will be the rule rather than the exception.

In recent years, many informative articles[2,3,4,5,6,7,8,9,10,11,12]

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* Read at the meeting of the Western Section of the American Laryngological, Rhinological, and Otological Society, Inc., Portland, Ore., February 6, 1954.

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have been written on the modified or conservative radical mastoidectomy. An additional paper on this subject might seem gratuitous were it not for the fact that there are still too many who feel that the modified radical is indicated in only an occasional case.

For example, as recently as 1950, McQuiston[13] stated, "In some cases in which the condition is confined to the epitympanic region, the patient may have useful hearing, indicating that the ossicular chain is intact and functioning. In such a case the modified radical mastoidectomy, or the atticomastoidectomy, is indicated; however, in our experience, there have been only a limited number of cases that have fallen into this category."

I must, of course, take issue with McQuiston's statements. For one thing, the presence of useful hearing *is not* proof of an intact ossicular chain; for another, the opportunities for some modification of the radical mastoidectomy *are not* rare and are not necessarily limited to only those cases where disease is confined to the epitympanic region, and where the ossicular chain is intact. To substantiate this opinion are the statistics presented in a previous paper of mine[14b] in which, in 64 consecutive cases treated surgically for chronic otorrhea, the modified radical (Bondy type) was done on 47 and the classical radical mastoidectomy on only 17; and, in Shambaugh and Derlacki's[14b] series of 62 cases, 36 had modified radicals and 26 had radical operations.

To obtain a dry ear is one of the goals in mastoid surgery but its importance is secondary to safety and function. Actually, thanks to Lempert,[15] who has shown us the way in improved technique and in the advantage of using magnifying lenses, achieving a dry ear in any modification of the radical has become no problem in most cases. Contrary to past beliefs, statistics[7,14b] show that most of the present day modified radical operations result in dry ears.

Indications for surgery will not be given, as they have been reviewed adequately elsewhere. This presentation is concerned with preserving or improving function while achieving safety.

What, then, is necessary to preserve or improve hearing? The most important single factor seems to be to maintain a freely movable stapes;[7,8] next, the round window area must be included in a sealed tympanic space connected with a patent Eustachian tube. The very best results, with some exceptions, are those which have an intact ossicular chain. Serviceable hearing can be obtained, however, with a sealed tympanic space in the absence of the incus and the head of the malleus,[4,6,11] but if this tympanic space becomes obliterated by eventual complete retraction of the tympanic membrane against the promontory, the good hearing level previously obtained may be lost.[11]

We must aim, then: *1.* To maintain the mobility of the stapes. *2.* To obtain a sealed tympanic space with continuity between the Eustachian tube and the round window area. *3.* To prevent obliteration of this sealed tympanic space.

To maintain stapes mobility, a few principles are as follows:

1. Avoid dislodging the stapes from the oval window by refraining from excessive manipulation about it such as may occur if an attempt is made to remove every bit of granulation tissue surrounding the stapes in its pelvis ovalis (fossula fenestrae vestibulae). It is not necessary to remove every vestige of granulation tissue as granulation tissue formation is reversible.

2. Avoid dislodging the stapes by not attempting to remove an enveloping cholesteatoma matrix if one is present.

3. Avoid removal of mucous membrane[7] in the pelvis ovalis near the fenestra, as this may cause excess scarring and stapes fixation.

4. Consider severing the stapedius muscle. According to House,[10] this may prevent a "tilting" of the stapes toward this muscle (when unopposed by the tensor tympani muscle through an intact ossicular chain) and may improve its mobility.

What can be done to prevent an obliteration of the tympanic space? Some suggestions are:

1. Preserve the ossicular chain when possible.

2. If the matrix of a cholesteatoma is sealing off a tympanic space, do not disturb the matrix.

3. If it is necessary to remove the incus and head of the malleus, leave behind some "tenting" structure. With the incus and the head of the malleus gone, tympanic membrane retraction may occur in an occasional case. If it happens in the only good ear of the patient, it could be a disaster. This "tenting-out" principle to prevent obliteration of the tympanic air space was demonstrated by:

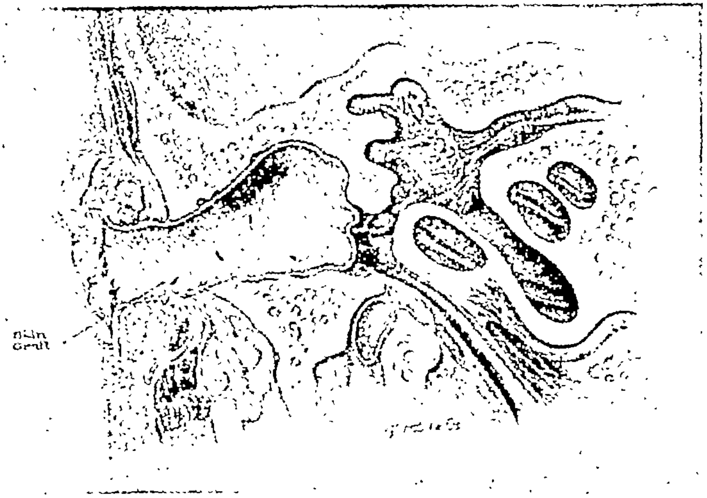

Fig. 1.

The bony ear drum, incus and malleus have been removed and a skin graft applied. If this technique is used, according to House, the hearing improvement is minimized because the graft lies over the stapes area and results in partial fixation. (In cases with which we are concerned, this does not seem to apply. In several patients the tympanic membrane closely enveloped the stapes and good hearing resulted.) (Courtesy Howard P. House: Congenital Ear Canal Atresia. The Laryngoscope, 63:925, 1953.)

a. House,[17] who, in a very fine paper on agenesis of the ear canal, described a method of preserving stapes mobility. He leaves intact the inferior bony plate (see Figs. 1 and 2), the bony "ear drum" present in these cases. This prevents the applied skin graft from resting upon the stapes* and the promontory, and results in a mobile stapes contained in a sealed tympanic space in continuity with the Eustachian tube. While there is no such bony plate in the ears with which we are concerned, a similar principle, such as described by Lempert, may be applied.

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* In the ears with which we are concerned, the approximation of the skin to the stapes is desirable providing that there remains a tympanic air space.

b. Lempert[6,7] suggested leaving intact the anterior bony "spike" (greater tympanic spine) and the posterior bony

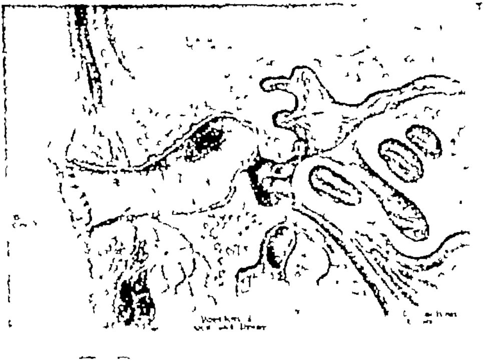

Fig. 2.

The procedure of choice leaves the bony ear drum largely intact. The deformed and fixed incus and malleus are removed through the smallest possible attic opening. A thin full-thickness skin graft is placed over this opening to act as a new ear drum creating a middle ear air space and avoiding partial stapes fixation. A maximum hearing improvement is attained by this technique. This illustration of House's is used to show how a bony projection will maintain a tympanic air space. (Courtesy Howard P. House. Congenital Ear Canal Atresia, The Laryngoscope, 63:926, 1953.)

"spike" (lesser tympanic spine) to help preserve the tympanic space and the epitympanic space, and to keep the tympanomeatal flap from adhering to the entire incudomalleolar joint. Lempert does not remove the incus and the head of the malleus in his modified radical operation, but for those who do, these spines may help to preserve the tympanic space. In this situation, when the incus and head of the malleus are removed, it is important to cover the space bounded medially by the medial attic wall and laterally by the spines and the tympanic membrane (the roof of the tympanum), with a skin flap or a skin graft.

c. If the incus has to be removed, it still may be possible to save the head of the malleus which will prevent complete retraction of the tympanic membrane against the promontory. I have tried this in but one case (see Case 7). The posterior portion of the tympanic membrane did retract to the promontory and to the capitulum of the stapes, but there was no retraction of the anterior half (see Fig. 16). The tympanic air space was maintained and excellent hearing resulted. This method deserves further trial.

TECHNICAL CONSIDERATIONS.

In this presentation, the term "modified radical mastoidectomy" is used in its broadest sense; that is, it includes any modification of the so-called classical radical mastoidectomy, and is not confined to the Heath or Bondy type of operation, although the Bondy is the one most often done. The Bondy operation, as referred to here, is the removal of the roof and posterior wall of the bony external auditory canal including the "bridge," the external attic wall, the opening up of the mastoid antrum and removal of all the bone external to it, and removing as much of the mastoid bone as may be required in any individual case. The management of the ossicles, the tympanic membrane and the membranous canal, varies with each situation.

BASIC TECHNIQUE.

I prefer the endaural technique, although any of these procedures can be done by the postauricular route if one so chooses. A modified Lempert approach is used with an S-shaped incision for the left ear and a reverse-S incision for the right, as described in a previous publication.[7] After the periosteum is elevated, a triangular piece of subcutaneous connective tissue is removed anteriorly in the region of the superior canal wall, to increase the exposure.

Before any bone work is done, the membranous canal is carefully separated from the bony canal in *all cases.* While one may have a previous plan, the final technique is not determinable until the area of pathology is exposed; therefore, one must not cut the membranous canal until he is certain of the

procedure to be done. This will allow the membranous canal skin to be used to the best possible advantage.

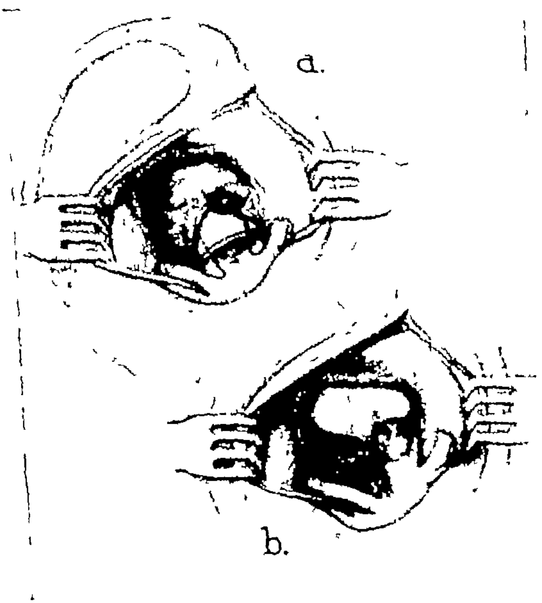

Fig 3

a A diagrammatic representation of the greater tympanic spine (1) and the lesser tympanic spine (2) Lempert[14] suggested that leaving these spines may be a way to prevent medial retraction of the tympanic membrane The lines drawn on the floor and sides of the membranous canal demonstrate the incisions to be made for a rotating flap that will cover and seal the tympanum. Note that the incision anteriorly is carried completely through the perforation, severing the anterior pedicle

b This illustration shows the flap in place attached by a thin posterior pedicle (The upper edge of the tympanic membrane is "freshened" before this flap is placed.) Primary union between the edges of the flap and the tympanic membrane may occur This type of flap may be used to cover and seal the tympanum either in the presence or in the absence of the ossicles

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The shortest approach to the mastoid antrum is through a

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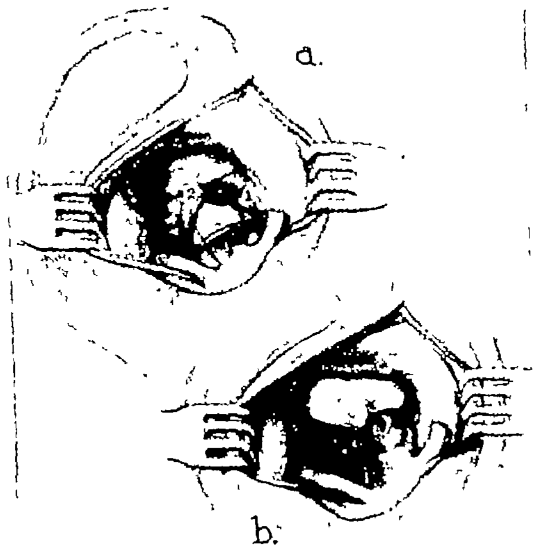

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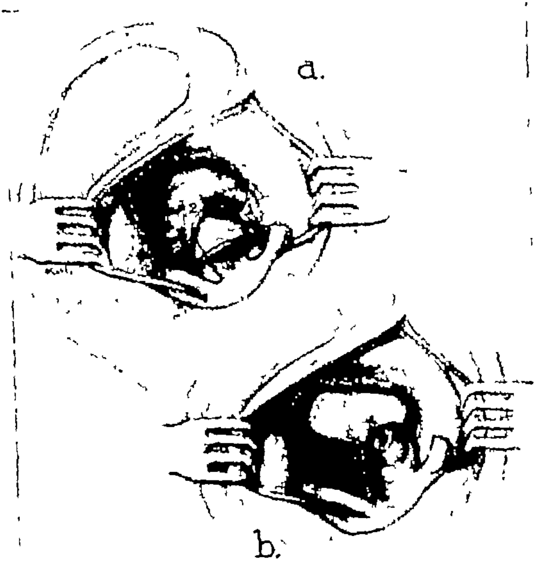

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point deep in the posterior bony canal wall, as suggested by Lempert,[15] although there is no objection to approaching the antrum via the cortex, if one so chooses. In a sclerotic mastoid, it is not necessary to develop a large mastoid bowl. In a pneumatic or partially pneumatic mastoid, one certainly should try to remove all cells possible. Predetermination of the anatomy by Xray is important. The posterior bony canal wall should be removed, and at its most external portion it should be leveled to the floor of the external auditory canal to prevent any pocketing in the mastoid bowl. I feel that the "bridge" should be removed in all cases.

The management of the membranous canal and the type of flap to be cut varies with the problem at hand.

SITUATIONS ENCOUNTERED.

I. Attic or attico-antral cholesteatoma with attic or posterosuperior-attic tympanic membrane perforation.

- *(a)* Technique when the cholesteatoma matrix is preserved.
- *(b)* Technique when the cholesteatoma matrix is removed.

II. Attic or posterosuperior-attic perforations where there is no cholesteatoma.

III. Large, non-marginal or marginal posterosuperior tympanic membrane perforations without cholesteatoma.

IV. When the tympanic membrane is absent and the middle ear is occupied by a cholesteatomatous mass.

V. Miscellaneous.

I(a) Attic or attico-antral cholesteatoma, with attic or posterosuperior-attic tympanic membrane perforation, when the cholesteatoma matrix is preserved:

Most of the cases of chronically discharging ears that require surgery have cholesteatomas, and a high percentage of these are from attic perforations (see Fig. 4) or posterosuperior marginal perforations, or a combination of both. Many of these have serviceable or close to serviceable hearing. It is expected, therefore, that they should have a good chance to maintain or improve their hearing. If the patient's middle ear cavity is sealed off by the matrix of the cholesteatoma, it is rare for the hearing to drop after surgery providing that the covering matrix is preserved and the middle ear cavity is undisturbed. Actually, many obtain an improvement in hearing, apparently as the result of the elimination of the middle ear congestion after the cholesteatoma is exteriorized. A number of these cases, preoperatively, have a serous otitis media which disappears after surgery, usually spontaneously, although occasionally drainage by myringotomy has to be done.

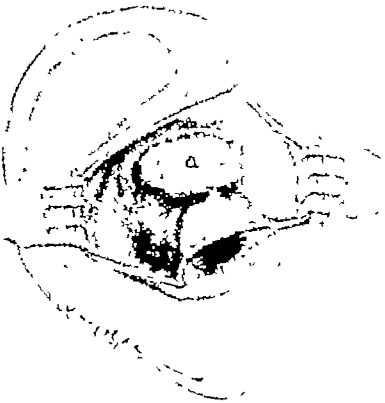

Fig 4

Diagrammatic representation of a cholesteatoma which in reality is a cyst "a" with a squamous cell epithelial lining on the inside, and a connective tissue layer on the outside. It is connected by a stalk to the perforation which in this case is in Shrapnell's membrane

When the matrix is saved, its perilabyrinthine extensions should be elevated and searched for any finger-like projections, which should be eliminated. It is not usual for the matrix to extend inferior to the labyrinth towards the mastoid tip, but when this does occur, this portion of the matrix should be removed and the underlying bone ground down in an attempt to eliminate nonvisible microscopic extensions that could appear later as recurrent cholesteatomatous cysts, especially since this area is to be covered with a skin flap. The overhang of the anterior buttress (greater tympanic spine) should be removed. The posterior overhang of the lesser tympanic spine should be removed only if it can be done without opening up the sealed-off tympanic space.

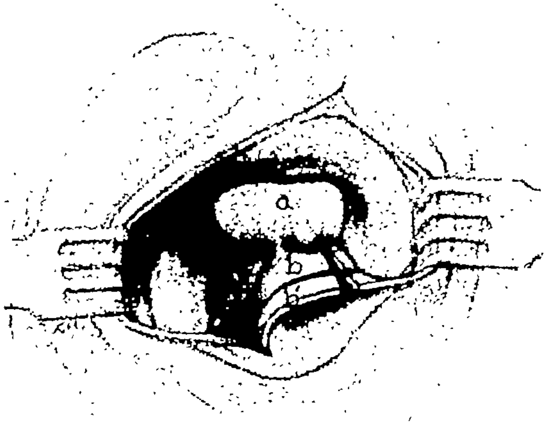

FIG. 5.

The outer portion of the cyst has been cut away and the inner layer (known as the matrix) is left undisturbed "a." Note that the incision in the long axis of the membranous canal is cut through the perforation, thus eliminating the perforation. The skin flaps "b" and "b-1" can now be reflected into the mastoid bowl.

The entire membranous canal wall, which now appears as a tube of skin, may be cut through its long axis superiorly down to and *through* the perforation, *thereby completely eliminating the perforation* (see Fig. 5). This forms a plastic flap, which can be reflected over the facial ridge to the mastoid bowl. For better conformity and maneuverability an external "cuff" should be separated from the main flap (see Fig. 6).

Should the preserved matrix of the medial attic wall be elevated to inspect the ossicles? That depends upon the preoperative hearing level. If the hearing is serviceable, especially if it has a drop of only 5 to 15 db, it is unwise, to disturb the matrix. If it is not disturbed, then how does one know the status of the underlying ossicles? One doesn't except for the presence or absence of active infection. When an infection is present, the matrix as a rule has a pinkish color, and is elevated by granulations which lie between it and the underlying structures. The elevation of this matrix by granulation tissue is quite obvious, and under the circumstances the matrix should be removed. Where there is no evidence of infection and the hearing is very good, the shape and continuity of the ossicles is of no importance.

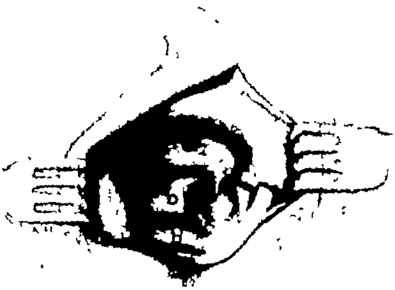

Fig 6

The completed operation "a" the cholesteatoma matrix left in situ (Note that it is covering the ossicles and is sealing off the tympanum) "b" and 'b-1' are the membranous canal skin flaps

There may be some wisdom in elevating the matrix to inspect the status of the ossicles, if the hearing is 50 or more db below threshold. In a case of Juers',[11] when this was done the malleus and incus were found to be fused and were removed; the matrix was replaced to seal off the tympanum. Following this, the hearing improved from a 50 db to a better than 20 db level. On the other hand, this isn't always necessary. In four of my cases, previously reported,[14b] with hearing losses from 59 to 69 db, the matrix was not elevated; yet, the hearing in each improved to a serviceable level.

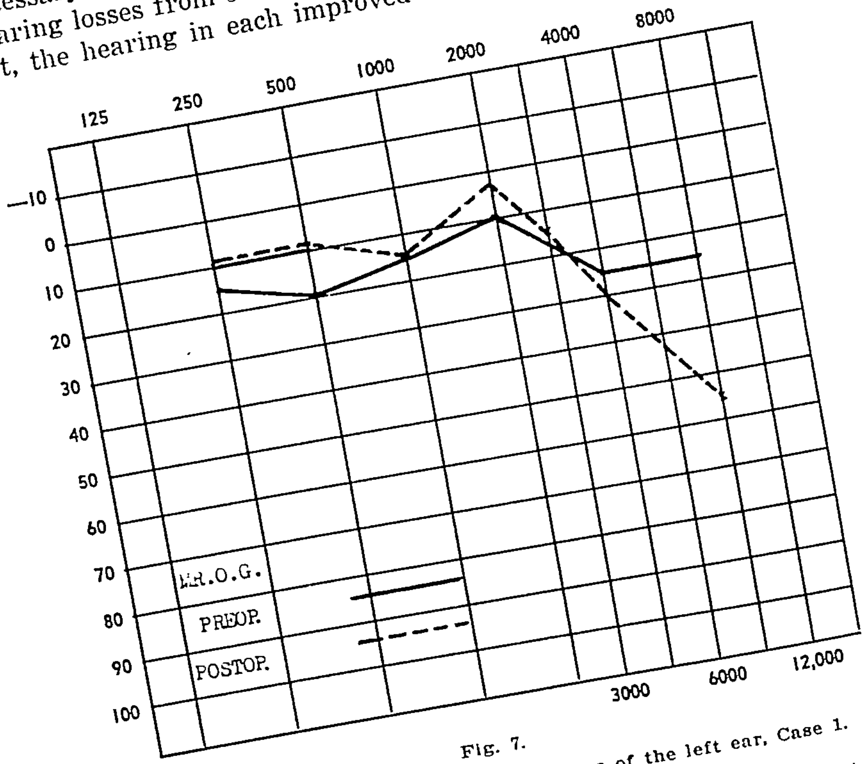

Fig. 7.

Preoperative and postoperative audiograms of the left ear, Case 1.

The plastic flap that has been reflected into the mastoid bowl is held in place with paraffin mesh gauze, which is left in place for about a week. The innermost layer of this gauze is marked with a black silk suture, which is brought out through the meatus. The preauricular portion of the incision is closed with silk.

There are instances in attic cholesteatoma where the matrix is medial to the incus and the head of the malleus. In those, cases, the incus and the head of the malleus may be removed. The matrix is undisturbed and is utilized to seal off the tympanum.

ILLUSTRATIVE CASE REPORTS.

Case 1. Mr. O. G., age 36. First seen April, 1947. Discharging right ear for 20 years. Discharging left ear, five years. Poor hearing in the right ear (58 db level), hearing within serviceable level (15 db level) in the left ear. Mucoid and muco-purulent drainage, right ear. Anterior attic perforation with a cholesteatoma plus a posterosuperior perforation in the left ear.

Surgery: Left, endaural, Bondy, modified radical mastoidectomy on September 15, 1948. An attic cholesteatoma extending to the antrum was found. The matrix in the antrum was covering granulation tissue, so this portion of the matrix was removed; that covering the tympanum and the ossicles was left *in situ*. Nothing was done to the postersuperior perforation through which could be seen fine granulation tissue, and this perforation eventually closed. The postoperative hearing level showed an improvement to 10 db (see Fig. 7). The last audiogram, done September 2, 1953, more than five years later, showed maintenance of the improvement. The ear is dry, and the ossicles are in bas relief, covered by a thin epithelium.

Comment: At surgery, would one dare manipulate the patient's middle ear structures and gamble on the future hearing in his only useful ear? Utilization and preservation of the matrix that sealed off the ossicles and the tympanum was the answer. One can expect that in this procedure there will be no further hearing loss. In cases previously reported, none that had serviceable hearing before operation lost this hearing when the matrix was saved, and some had further hearing improvement; also, some patients who were below the serviceable level reached the serviceable level after surgery.

Of the cholesteatomas encountered the attico-antral one, as present in this patient, is found most often,[14b] It is obvious, therefore, that *this technique of preserving the matrix is one of the most valuable in conservation of hearing.*

Case 2. Mrs. D. McC., 51. First seen March 23, 1953. Complained of progressive loss of hearing in her only good ear, the left, for about ten months without any history of ear discharge. Deafness in her right ear was present following a radical mastoidectomy done elsewhere five years previously. There was no perforation in the tympanic membrane or in Shrapnell's membrane of the left ear, but through Shrapnell's membrane could be seen a whitish, semilunar mass. It was apparent that this was a walled-off attic cholesteatoma, and that its progressive expansion was destroying the hearing in that ear. A left, endaural, modified radical mastoidectomy of the Bondy type was done on April 17, 1953. The attic and mastoid antrum were occupied by a bean-shaped cholesteatoma. The matrix was medial to the incus and head of the malleus and had sealed off the tympanic cavity below. The incus was ankylosed to the fossa incudus and came off with the "bridge." The atrophic head of the malleus was amputated. The matrix was left *in situ*.

The postoperative audiogram (see Fig. 8) shows an improvement in hearing for the conversational range, and a slight loss at 4000 cycles per second. The ear is dry.

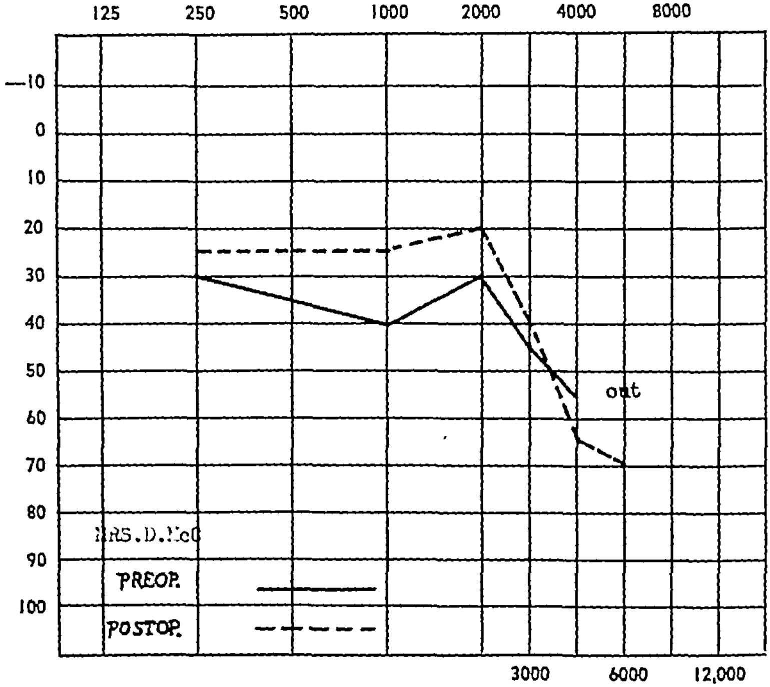

Fig. 8.

Preoperative and poostoperative audiograms of the left ear, Case 2.

Comment: This case demonstrates again the value of not "overmanipulating." One must take advantage of nature's having sealed off the middle ear with the cholesteatoma matrix, especially when this situation is in the only ear with hearing.

This demonstrates also that one must be constantly aware of the possibility of middle ear or mastoid pathology without a tympanic membrane perforation.

Three situations were encountered where it was necessary to remove part of the matrix in the epitympanum while it was possible and desirable to save the most anterior portion. This left the short process and part of the body of the incus uncovered (see Fig. 9). A flap was made to cover the bared incus and the uncovered portion of the tympanum as follows: a Lempert type of tympanomeatal flap was developed in the usual manner. When this was done the anterior auditory canal wall attachment was severed in the direction of and through the attic perforation, thus eliminating the perforation (see Fig. 10). This left the flap with a single posterior pedicle, so that it could be rotated posteriorly sufficiently to permit the anterior cut end to lie on the uncovered incus and tympanum with its free edge closely approximating the free edge of the remaining attic portion of the matrix (see Fig. 11). It was held in place with paraffin mesh gauze, which was removed in one week. *Primary union* occurred at the

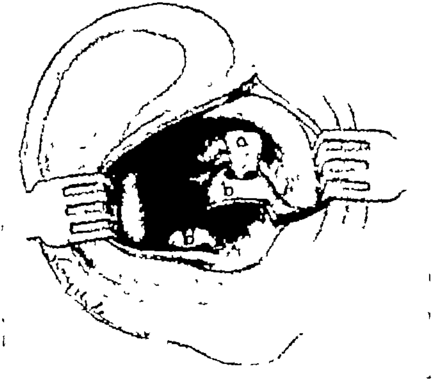

Fig 9

"a" Cholesteatoma matrix covering the head of the malleus, part of the body of the incus, and part of the tympanum It was necessary to remove the posterior portion of the matrix, leaving the short process and part of the body of the incus uncovered An external cuff "b-1" of the membranous canal has been reflected into the mastoid bowl (Cases 3, 4, and 5) margins in each case and the hearing was maintained or improved in each instance.

Case 3. Miss M. B., a nurse, age 33. First seen September 5, 1952. No history of running ears. A perforation of the right tympanic membrane was noticed during a routine physical examination two years previously.

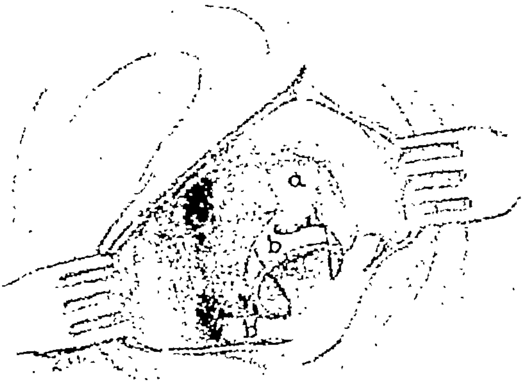

Fig. 10.

The solid and broken lines starting from the floor of the membranous canal indicate the incisions to be made for the flap desired. Note that the anterior incision is carried through the pedicle and through the perforation, thus eliminating the perforation.

She complained of moderate deafness which became almost complete during an upper respiratory infection. Initial examination showed very thickened, retracted tympanic membranes with a bilateral secretory otitis media. Shrapnell's membrane of the right ear was retracted flat against the medial attic wall with an opening between this and the external attic wall into the mastoid antrum. There was definite evidence of a cholesteatoma. The left ear showed attic retraction but no perforation.

Surgery: October 31, 1952. Right, endaural, Bondy type of modified radical mastoidectomy. A cholesteatoma was found extending into the mastoid antrum with a pinkish raised matrix with obvious granulation tissue between matrix and bone. The matrix, therefore, was removed to the retracted Shrapnell's membrane. This exposed the short process and part of the body of the incus. The incus and mastoid antrum were covered by a modified tympanomeatal flap just described (see Figs. 9, 10, 11).

Result: Primary union between the margins of the flap and the retracted Shrapnell's membrane. Ear dry. Preoperative hearing level of 40 db, postoperative 35 db, not a significant improvement, but there was no further loss. Preoperative bone conduction level was 20 db.

Case 4.[*] Mrs. L--l, age 31. Bilateral suppurative otitis media of many years' duration. Anterior attic perforation with cholesteatoma in her right ear, and a posterior-inferior tympanic membrane perforation in the left. Preoperative hearing, right ear, 21.6 db, 40 db for the left. And endaural, right, Bondy type modified radical mastoidectomy was done on the right ear, February 2, 1953. There was an attic cholesteatoma extending to the antrum. It was necessary to remove the matrix in the antrum to a point anterior to the incus, thus exposing the short process and the body of the incus. Granulations under the incus and encompassing the stapes were noted, but these were not disturbed. The same type flap was used to cover the incus as in Case 3 (see Fig. 11). The point of approximation healed by primary union. About two weeks postoperatively a small one-millimeter perforation developed in the posterior-inferior quadrant of the pars tensa of the tympanic membrane. The middle ear drained through this perforation, but drainage ceased after two weeks and the perforation closed.

Result: a dry ear and an improvement in hearing from 21.6 db to a 16.6 db level (see Fig. 12).

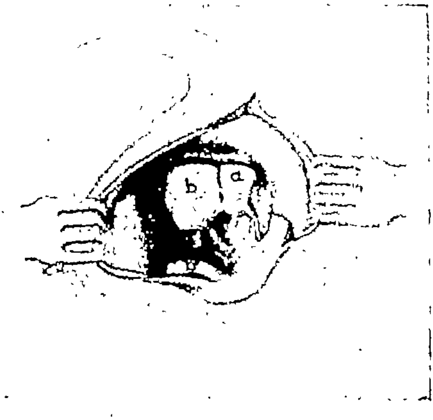

Fig. 11.

The completed operation. Note how the skin flap "b" covers the incus and the previously uncovered tympanum. It is closely approximated to the edge of the undisturbed matrix "a." Primary union occurred between these edges in Cases 3, 4, and 5.

Comment: This was an "experimental" procedure to determine what would happen if a middle ear containing granulation tissue were sealed off by a flap, and if primary union between the edges of the flap and the free margin of the matrix could occur under the circumstances. It was apparent that the tympanic membrane perforation occurred from the accumulation of middle ear exudate from the granulation tissue. When the main "mass" of the disease was removed surgically, the granulations resolved, the discharge ceased and the perforation healed. Primary union of the "edges" was not inhibited by the underlying granulation tissue. Conservatism "paid-off" in the only good ear of this patient.

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[*] Cases 4, 5, 6 are from the Letterman Army Hospital. They are presented through the courtesy of Col. Byron G. McKibben, Chief of the Ear, Nose and Throat Service, and the courtesy of the Surgeon General of the Army.

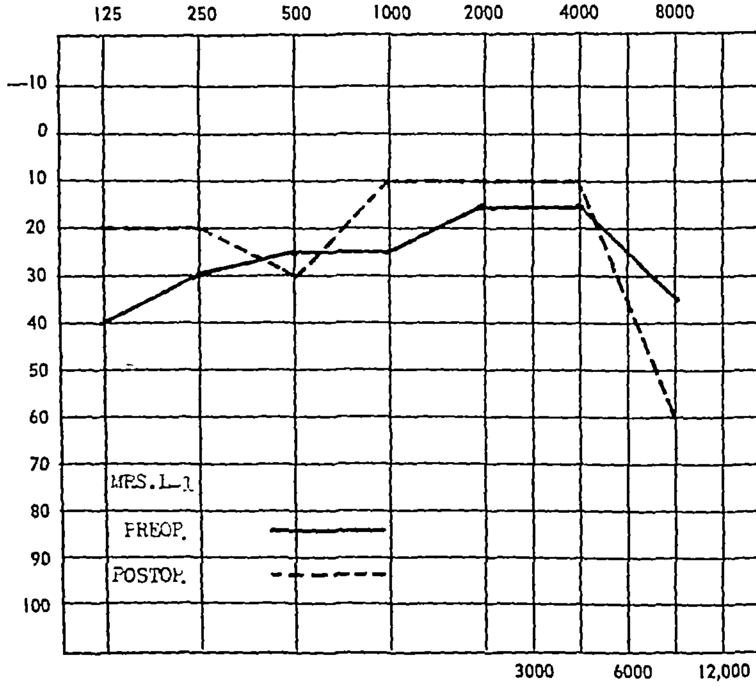

Fig. 12.

Preoperative and postoperative audiograms of the right ear of Case 4.

Case 5. L.-2, a 14-year-old boy. Draining left ear for eight years. Posterosuperior perforation of the pars tensa continuous with a posterior attic perforation with an attic cholesteatoma.

Surgery: July 20, 1953, a left, endaural, Bondy, modified radical mastoidectomy. The mastoid was moderately pneumatic and the cells and mastoid antrum were filled with granulation tissue, all of which was removed. The attic cholesteatoma was small, and when it was externalized part of the incus was left uncovered A flap as in Cases 3 4 was fashioned to cover the antrum and the uncovered incus to the edge of the matrix Primary union between the edges occurred Result a dry ear and an improvement in hearing in from 1[illegible] db to 11 6 db

I(b) Attic, or attico-antral cholesteatoma, with attic or posterosuperior-attic tympanic membrane perforations when the cholesteatoma matrix is removed:

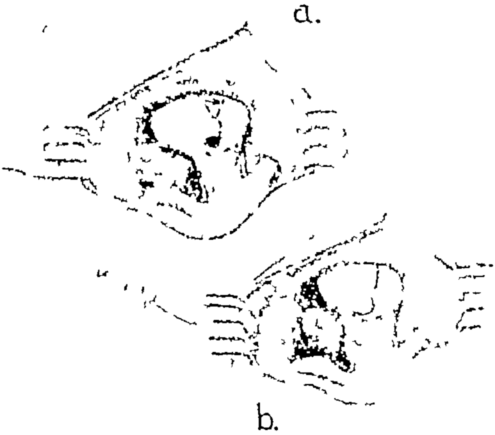

Fig 13

a If a typical Lempert type tympanomeatal flap is used in the presence of an attic perforation there will remain a perforation within this flap (Fig a) The broken lines suggest a method of incision to rotate the posterior half of the flap forward and downward to eliminate the perforation. (The margins of the perforation are freshened before this is done) Fig b—The perforation is closed.

There are some[15] who do not believe in saving any portion of the matrix. Of these, there are those who carefully remove the matrix and try to leave the ossicular chain intact if it is apparent that the ossicles are not grossly involved; others[4] remove the matrix and routinely take out the incus and the head of the malleus. In both instances the tympanum has been covered with a Lempert type of tympanomeatal flap. This flap, under the circumstances, will always have an attic fistula, since the perforation existed preoperatively in the attic or the posterosuperior portion of the tympanic membrane (see Fig. 13-a). Lathrop,[4] who permits these to close spontaneously, admits that approximately 50 per cent of these perforations remain open with approximately a 25 per cent incidence of constant or intermittent drainage.[19] These unhealed attic fistulas are always potential sources of new cholesteatoma. Lathrop has never seen this happen,[10] but Kenneth Day[20] stated that he has observed this sequela in two cases.

If one chooses to use a tympanomeatal flap with a perforation, then one should try to eliminate the perforation by:

1. Splitting the flap from the perforation to the superior edge of the flap and freshening the margins of the perforation; then approximate the edges by gentle teasing together and hold them in place with paraffin mesh gauze (see Fig 13b). In addition to closing the perforation, this will seal the tympanum.
2. According to Martin,[21] one should try to mobilize the tympanic membrane sufficiently to permit the perforated portion of the tympanomeatal flap to be "plastered" against the medial attic wall just above the prominence of the tympanic portion of the facial nerve. An incision is then made in the flap between the perforation and upper edge and the cut margins approximated, as just described.
3. A further method, and the one I prefer, is to develop a Lempert type of tympanomeatal flap, severing the anterior attachment in a manner so that the incision reaches the perforation, thus eliminating the perforation. This leaves a very mobile flap on a very small posterior pedicle which can now be rotated to cover the tympanum. This is similar to the flap used in Cases 3, 4, and 5 except that, instead of approximating the anterior cut end to the margin of an epitympanic matrix, it is rotated anteriorly sufficiently to completely seal the tympanum (see

Fig. 3-b). The margins of the perforation should be freshened before approximating the flap.

Case 6 Mrs L.-3, age 50 years, with drainage from her right ear for three years. The pars tensa of the tympanic membrane was pulled over to the promontory with a perforation involving Shrapnell's membrane This perforation was part of a space between the retracted pars tensa and the external attic wall leading to the mastoid antrum

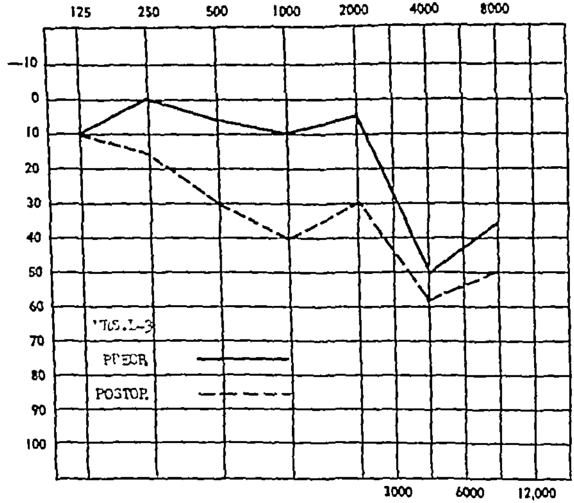

Fig 14

Preoperative and postoperative audiograms of the right ear of Case 6

Surgery January 5, 1953, an endaural, Bondy type, modified radical mastoidectomy An attic cholesteatoma was present. The matrix was entwined between and around the incus and the head of the malleus. It was necessary to remove the incus and the head of the malleus. When this was done, the matrix did not adequately cover the tympanum so the matrix was removed A flap was developed, as described in the previous paragraph. Primary union occurred between the lateral border of this flap and the upper margin of the tympanic membrane

Result A dry ear and a considerable loss in hearing from a 6.6 db to a 33.3 db level (see Fig 14)

Comment: This case demonstrates how the tympanum can be sealed with a non-perforation-containing flap. It demonstrates also that one can't always be sure of retaining hearing when the incus and head of the malleus are removed. It further shows that a small tympanic air space may be inadequate in such a situation. The tympanic membrane was retracted to the promontory before surgery; postoperative Eustachian tube inflations in an attempt to separate the tympanic membrane from the promontory were not successful. Possibly the ossicles should have been left, since the preoperative hearing was so excellent.

4. Another way to seal the tympanum is by the use of a thin skin graft, either in the presence or absence of the incus and head of the malleus. Meltzer[22] suggested that this graft extend one millimeter over the upper edge of the tympanum. When the ossicles are not present, mobilization of the tympanic membrane will enhance the chance of a "take." Since the membranous canal is not utilized for this purpose* here, it may be cut and reflected into the mastoid bowl, as in the situation where the attic cholesteatoma matrix is preserved (see Figs. 6-b1 and b2).

5. A further method of the management of the tympanic air space is that which has been used successfully by Robert C. Martin. Martin[21] feels that a sealed tympanic air space is necessary for a good hearing result, but he thinks that this space should be very small. This conclusion was reached by his observation that poor hearing resulted in certain patients (not his own cases), each of whom had his incus inadvertently removed when the simple mastoidectomy was performed. Martin believes this poor hearing is due to too much tympanic air space lessening the transmission of sound to the stapes. Actually, there is more than the size of the tympanic air space, *per se*, to be considered here. There are the air-filled connections of the tympanum, such as the aditus and mastoid antrum, which may act as "dilution" factors in the transmission of sound to the stapes.

In a few cases where he had to remove the incus and the head of the malleus, Martin severed the tensor tympani muscle and teased the handle of the malleus out of its tympanic membrane "casing." This "skinning" of the malleus is started just below the short process, with the head of the malleus firmly fixed with a forceps. The tympanic membrane and connective tissues are carefully teased from the outer surface of the handle of the malleus. The mucous membrane apparently comes out with the handle of the malleus and leaves a raw

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* To seal the tympanum.

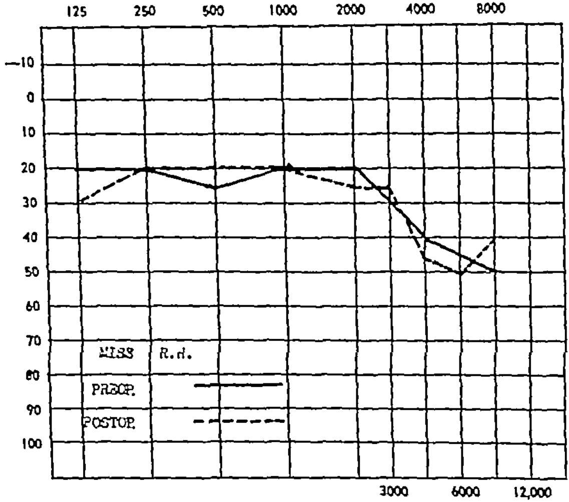

Fig 15

Preoperative and postoperative audiograms of the right ear of Dr Robert C. Martin's Case

surface where the tympanic membrane may attach itself to the promontory. The upper two-fifths of the tympanic membrane is elevated from the annulus; when the upper annulus and lesser tympanic spines are removed, the tympanic membrane, when replaced, is permitted to fall against the stapes and against the region of the tympanic portion of facial nerve and the processus cochleariformis. Anteriorly, the tympanic membrane is allowed to fall flush against the opening of the

Eustachian tube and is held firmly against the promontory by a pack. In one case the tympanic membrane was mobilized sufficiently to be moved forward over the opening of the Eustachian tube. The stapes area and the facial ridge area were covered with two very thin separate skin grafts, which were held in place with paraffin mesh gauze. Eventually a small sealed tympanic air space developed and a good hearing result was obtained (see Fig. 15).

II. Attic or posterosuperior attic perforation where there is no cholesteatoma:

The procedures here are essentially the same as for *I-b*, where the cholesteatoma matrix is removed, since removal of the matrix creates an identical situation.

III. Large marginal or non-marginal posterosuperior tympanic membrane perforation without cholesteatoma:

In the past, some cases in this category were treated surgically by an extensive cortical (simple) mastoidectomy. With the current techniques better results, both as to hearing and freedom from discharge, can be obtained by the modified radical operation due to better exposure, more complete eradication of the disease and elimination of "dead" space; and, by the complete removal of the external attic wall and "bridge," a better "draping" of the skin in continuity with the tympanic membrane onto the medial attic wall will occur. This increases the chances for closure of the perforation and the development of a sealed tympanic space with better hearing.

Should the perforation fail to close after the ear is dry, closure may be accomplished by the methods suggested by House[23] and Martin:[21]

a. (Method suggested by House[23]). Freshen the margins of the perforation and make three equally spaced stellate incisions from its edges to the annulus. Strip the mucosa of the promontory. Cut a thin, full-thickness skin graft from the post auricular area and apply to the perforation and denuded promontory. Hold the graft in place with a cotton ball pack.

b. (Method suggested by Martin[21]). Lift the posterior portion of the tympanic membrane from the annulus by incising enough of the canal wall skin to permit this to be done. Remove the upper-posterior portion of the annulus and "roughen" the adjacent bone of the posterior canal wall. Freshen the edges of the perforation and make an incision from the perforation through the remainder of the tympanic membrane posteriorly. This allows the cut segments of the tympanic membrane to fall toward the medial wall where mucosa has been "roughened." Apply a thin, split-thickness skin graft large enough to cover the split perforation and the roughened posterior bony canal wall. Use a small pack for pressure.

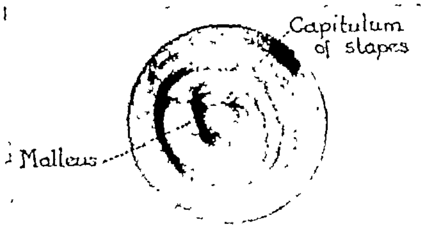

FIG. 16

Appearance of the healed tympanic membrane in Case 7. The capitulum of the stapes is closely enveloped by the new membrane of the healed perforation. Its retraction toward the medial wall is evident. The anterior one-half of the tympanic membrane has not retracted. There is an adequate sealed tympanic air space and the stapes is sufficiently mobile for good hearing.

CASE REPORTS.

Case 7. Miss N. C., age 14. First seen February 5, 1949, with a discharging left ear and a posterosuperior marginal perforation of the tympanic membrane. Hearing at that time, 45 db left ear, normal in right. Mastoid Xrays then showed a chronic left mastoiditis without evidence of bone erosion. The ear became dry with local treatment. Seen again July 20, 1950, for recurrence of the drainage and dizziness following swimming. Perforation was about the same. The discharge stopped with local treatment. Was not seen again until June 23, 1953. Returned then because of persistent drainage in the left ear for about six months. This discharge was foul smelling. Examination showed that the posterosuperior perforation had increased in size. Re-Xrays of mastoid revealed "the left mastoid antrum is definitely sclerotic and there is a 7 mm. in diameter radiolucency in the mid portion of this antrum. This is presumably due to a cholesteatoma." There was further loss of hearing to 50 db.

Surgery: A Bondy type of endaural, modified radical mastoidectomy with a Lempert tympanomeatal flap was done on July 24, 1953. Considerable bony erosion was noted in the mastoid antrum and in the solid angle. No cholesteatoma was found. The entire mastoid, the middle ear, including the pelvis ovalis and the stapes, contained abundant granulation tissue. The incus was deformed with some loss of both long and short processes. The incus was removed but the head of the malleus was left *in situ* (experimental, in an attempt to prevent medial retraction of the tympanic membrane). All granulation tissue in the mastoid and epitympanum and as much as possible in the pelvis ovalis was removed, but that surrounding the stapes was left undisturbed. It was felt that the granulation tissue might disappear and that the tympanic membrane perforation, although large, might close under treatment. There was remarkably little drainage postoperatively. By September 8, 1953, all purulent drainage had disappeared. At various intervals the perforation edges were cauterized with trichloracetic acid and a Cargile membrane patch was applied. On December 9 the perforation had healed. The new membrane closely hugged the capitulum of the stapes, but the anterior half of the tympanic membrane did not retract against the medial wall (see Fig. 16). The last audiogram on January 28, 1954, showed an 18 db level—an over-all improvement of 32 db (see Fig. 17).

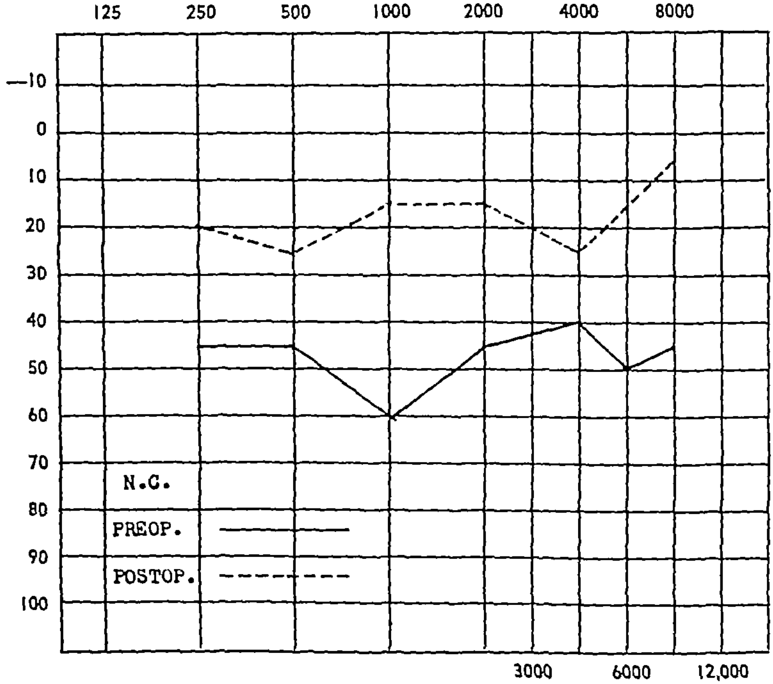

Fig. 17.

Preoperative and postoperative audiograms of the left ear of Case 7.

Comment: This is a very instructive case as it shows:

1. That granulation tissue formation is reversible.

2. That a large tympanic membrane perforation, even though marginal,* can be closed.

3. That pressure of the new tympanic membrane against the capitulum of the stapes is no deterrent, but probably an aid to hearing as long as the sealed tympanic air space is adequate.

4. That the head of the malleus can act as a protuberance to "tent-out" the tympanic membrane sufficiently to prevent its complete retraction against the promontory.

Case 8. R. St. P., 16-year-old male. Discharging left ear since the age of 5. At one time the right ear had discharged also, but it was now dry. The hearing level was 33.3 db for the right ear and 50 db for the left. Considerable purulent drainage was present in the ear canal, and a large middle ear polyp was projecting through the perforation of the tympanic membrane. This large perforation involved almost the entire drum membrane except for a complete margin at the periphery.

Surgery: December 26, 1952, a Bondy type, endaural, modified radical mastoidectomy. Considerable granulation tissue was found throughout the mastoid, the epitympanum, and in the middle ear. The incus and malleus were not disturbed, and the granulation tissue adhering to these was not touched. The middle ear granulation tissue that could be reached superficially through the perforation was removed. A Lempert type tympanomeatal flap was made to cover the incus and the head of the malleus. Considerable purulent drainage was present for a long time after surgery. It was not until August, 1953, that the discharge lessened; also, at this time, the perforation began to diminish in size. In September, 1953, the ear was dry. Trichloracetic acid was applied to the margins of the perforation on several occasions, and on October 21, a Cargile membrane patch was applied. It was re-applied on November 12 and in December, when the patch was removed, the perforation was seen to be completely healed An audiogram done on December 9, 1953, showed an improvement from a 50 db to a 12 db level.

Comment: This case, too, shows that granulation tissue formation is reversible, and that a large tympanic membrane perforation can be closed with this procedure. This was much more difficult to control, postoperatively, than the previous case, but with persistence, control was obtained and the perforation closed with improvement in hearing. This was important for this patient as, preoperatively, both ears were below the serviceable level. Now the left ear has the better hearing.

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* It is not possible to close a marginal tympanic perforation in a non-operated ear.[illegible] The difference, apparently, is brought about by the surgical removal of the adjacent bone (the "bridge" and tympanic sulcus). It is not known, as yet, whether or not a skin graft, as suggested by House,[illegible] will close a marginal perforation in an unoperated ear.

IV. When the tympanic membrane is absent and the middle ear is occupied by a cholesteatoma:

In cases falling in this category, the surgical technique differs from the classical radical mastoidectomy only in that the matrix, which is firmly approximating the stapes, the promontory and the Eustachian tube orifice, is not disturbed. The hearing improvement which occurred in the two cases herein reported is difficult to explain, because there is no demonstrable tympanic air space.* After complete healing, the matrix was seen to be changed from a "stiff" thick, scaly structure to a clean, pink, thin, pliable skin. It is probable that this improvement in mobility, transferred to the stapes, may account for the hearing improvement.

CASE REPORTS.

Case 9. G. P., a 14-year-old male. Draining left ear for ten years. The left external auditory canal was only a "slit," the result of the large cholesteatoma having pressed the posterior portion of the membranous canal forward. Audiogram: average db level for 250 to 8,000: 28.3; average for conversational range, 46.6 db.

Surgery: January 9, 1953. Findings: A very large cholesteatoma which had eroded most of the posterior bony canal wall and part of the bony covering of the vertical portion of the facial nerve. The matrix was continuous with skin of the floor of the external auditory canal; hugged the promonotory covering the stapes and the Eustachian tube, and extended to the medial attic wall. External to this were remnants of the incus and malleus and a small bit of upper tympanic membrane. Endaural technique: All the matrix was removed except that covering the stapes, the round window, the promontory and the orifice of the Eustachian tube and that covering the upper vertical portion of the facial nerve. Attempts to separate the matrix from the nerve produced severe facial contractions. The incus, malleus, tympanic membrane remnants and tensor tympani muscle were removed. A flap of skin from the membranous canal was reflected into the mastoid bowl. Result: Ear dry. Postoperative audiogram, December 23, 1953: over-all level, 23.3 db; conversational range, 36. 6 db (see Fig. 18).

Comment: There is no evidence of a tympanic air space here, and no apparent one could be produced by catheterization and inflation of the Eustachian tube. The bizarre, unexplainable preoperative pattern of the audiogram was maintained with some improvement after surgery. It is doubtful that this result would have been obtained had the matrix been removed from the stapes and the hypotympanum.

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* McNaught[21] believes that an undemonstrable hypotympanic air space between the Eustachian tube and the round window is present in these cases.

Case 10. N. L., an 11-year-old boy. Discharging left ear since infancy. Hearing loss, left ear, 80 db; right ear, 7 db. Middle ear occupied by a cholesteatoma.

Surgery: September 18, 1953. Endaural technique. The matrix of the cholesteatoma extended from a small inferior rim of tympanic membrane to the medial wall covering stapes, the promontory and the Eustachian tube orifice, and extended to the attic and the mastoid antrum. There were remnants of the incus, malleus and Shrapnell's membrane, which were external to the cholesteatoma. The matrix in the antrum covered granulations and was removed. The matrix, which covered the stapes, the round window, the promontory and the Eustachian tube was not disturbed. The membranous canal was cut to reflect a flap into the mastoid bowl. Result: Ear dry on November 23, 1953. An audiogram, November 24, 1953, showed that the preoperative level of 80 db had improved to 48 decibels.

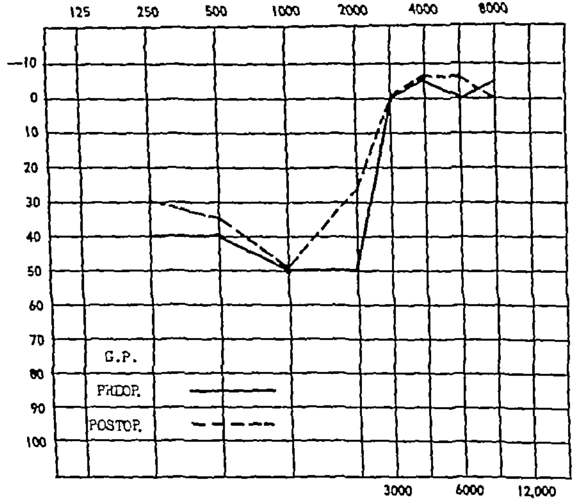

Fig. 18.

Preoperative and postoperative audiograms of the left ear of Case 8.

Comment: A 48 db threshold is far from the serviceable level, but if a person has bilateral deafness, he would be grateful for an improvement from an 80 to a 48 db level.

V. Miscellaneous:

This heading was included in order to discuss two cases that resulted in remarkable hearing improvement following the classical radical operation. This improvement, not anticipated, suggests that a planned improvement in hearing in the "complete" radical mastoidectomy may be possible.

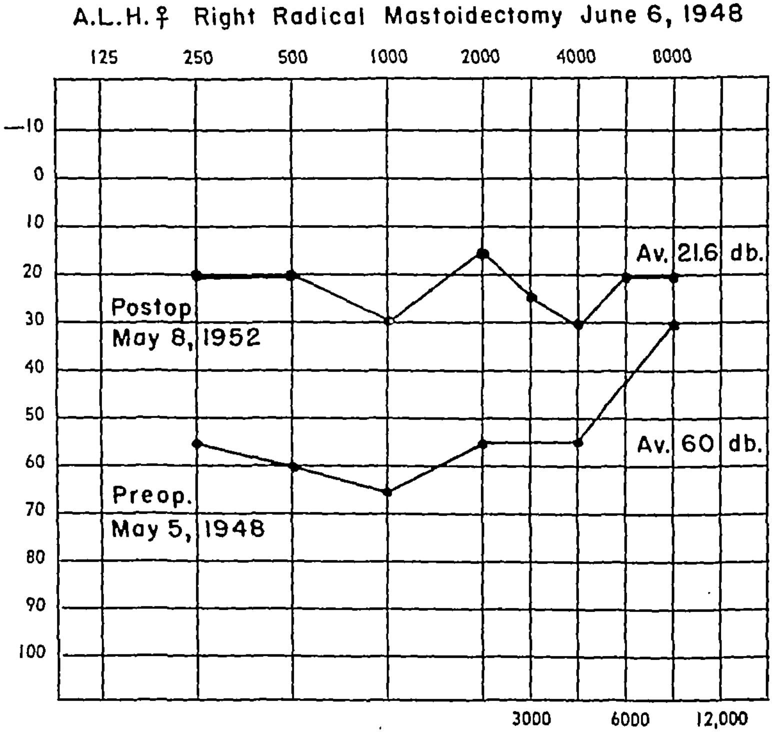

Fig. 19.

Preoperative and postoperative audiograms of the right ear of Case 11.

Case 11. Miss A. L. H., age 19. Discharging right ear since infancy. Had some type of radical operation elsewhere in 1943, and on two occasions later had incision and drainage of postauricular abscesses. Blood-tinged, purulent, discharge was present in the canal. Granulation tissue was present in the middle ear, the epitympanum, and in the mastoid antrum. At surgery on June 4, 1948, (endaural technique) there was no evidence of an incus or malleus, and the mastoid bowl and external auditory canal were united into a common cavity. The stapes was covered with a layer of thin scar tissue, which was not disturbed, but the remainder of the middle ear, the epitympanum and mastoid were thoroughly cleaned and a skin flap was laid into the mastoid bowl. Postoperative epithelization occurred rapidly. A pseudo-tympanic membrane (unplanned) developed. The preoperative hearing level of 60 db improved to 21.6 db (see Fig. 19).

Comment: This remarkable hearing improvement in a classical radical mastoidectomy was not a planned one. Why, then, did it occur? The probable answer is that the pseudodrum membrane formed a sealed tympanic space in continuity with the Eustachian tube and round window area and that the stapes, with its thin scar tissue covering, was sufficiently mobile to transmit sound. It is this case, and the following one, that suggest that we should attempt a planned hearing improvement even in the classical radical mastoidectomy.

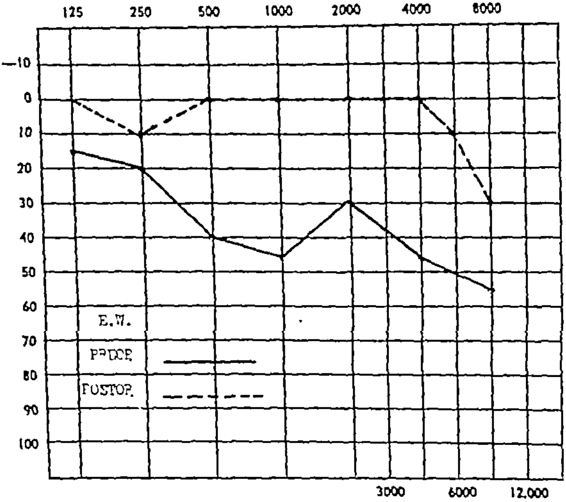

Fig. 20

Preoperative and postoperative audiograms of the right ear of Case 12

Case 12 E. W., a 20-year-old female Discharging right ear for 12 years Complained of pain in the right mastoid area, dizziness and occasional double vision Examination: purulent discharge in her right ear with a large, non-marginal posterosuperior perforation, there was no gross ocular muscle paresis, but an examination by an eye consultant showed minimal right external ocular muscle palsy. The neurological consultant found no evidence to indicate a brain abscess The electroencephalogram suggested local meningeal irritation Audiogram: 38 db level for the right ear

Surgery: April 7, 1952 Findings: granulation tissue filled the middle ear, epitympanum and mastoid; there was no evidence of an incus or head of the malleus. Endaural technique, classical radical procedure, but the granulations about the stapes were not disturbed. A flap of skin from the membranous canal was reflected into the mastoid bowl. The remaining structures were left bare. Epithelization was rapid and a pseudo-drum membrane (unplanned) developed. Result: Ear dry. Preoperative level of 38 db improved to 3.3 db level (see Fig. 20).

Comment: Like Case 11, this was an unplanned fine hearing result. The new pseudo-tympanic membrane hugs the capitulum of the stapes, but there is an obvious adequate sealed tympanic air space in continuity with the Eustachian tube. Not touching the granulations about the stapes at surgery proved to be a proper caution, as the granulation tissue dissolved and the stapes retained complete mobility.

DISCUSSION.

It is not the intent of this presentation to renew the controversy[14] as to whether or not it is safe to preserve the matrix of a cholesteatoma, but the subject can not be ignored since most of the cases that require surgery have cholesteatoma, and the over-all best hearing results obtained are in those in which the matrix is preserved. One can and must preserve the matrix without any qualms. There are situations, of course, wherein it is unwise to preserve the matrix and accept the penalty of a possible hearing loss. These will be discussed later.

Among those who oppose preserving the matrix is Walsh,[25] who stated, "If you leave cholesteatoma in place without destroying the matrix entirely, you may give the patient a dry ear for a while, but he is 'sitting on a keg of dynamite'." Walsh goes on to mention the many things that might happen to such a patient. Actually, the complications which he enumerates have occurred in an unoperated cholesteatoma, but they do not happen in a postsurgical case when the cholesteatoma is exteriorized and the cavity is treated until it is dry. Wetness in *any* kind of postoperative mastoid cavity is a potential source of trouble. Those of us who have been preserving the matrix for many years, Juers,[11] McNaught, Shambaugh, Shannon, and others,[14b] have never seen a sequela from this procedure that even faintly resembles a "keg of dynamite."

Williams[20] believes that in about 10 per cent of the cases in which cholesteatoma matrix is preserved the cavity fills with cholesteatomatous debris. That has not been the experience of others[14b] who have used this technique. We find it difficult to differentiate between the cavity in which the matrix has been saved and any other cavity, be it fenestration or mastoid.

For those of you who have not practiced saving the matrix and who may be interested in doing so, the following rules may be helpful:

WHEN AND WHY THE MATRIX SHOULD BE PRESERVED:

Saving the matrix is recommended for the following reasons:

1. The layer of the matrix that is to be externalized is squamous epithelium and serves to obtain a completely epidermatized mastoid cavity and a dry ear.
2. In sclerotic mastoids there will be less chance for the formation of recurrent cholesteatomatous cysts.*
3. In general, more conservative mastoid surgery will be done with an aim towards better conservation of hearing. In the attic type of cholesteatoma, maintenance or even improvement of hearing is almost certain, since preserving the matrix leaves the middle ear structures undisturbed.
4. In the presence of certain labyrinthine fistulas, the matrix will act as a barrier against infection entering the labyrinth.
5. In exposed dura mater in any area including lateral sinus, where there is no clinical evidence of infection, locally or intracranially, the matrix may be firmly adherent. Leaving the matrix will prevent surgical trauma to the dura. The presence of disease in these areas usually will be evidenced by granulations, or a pinkish color of the matrix. In these situations, the matrix may be removed readily.
6. In facial nerve paralysis caused from the pressure ef-

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* Reasons for this have been described in a previous paper [14b]

fects of a cholesteatoma, and in which the function may return when the pressure is released, an attempt to remove an adherent matrix from the nerve may produce some injury to the nerve; however, when there is bone necrosis about the nerve, as evidenced by granulation tissue interspersed with matrix, the matrix should be removed.

7. In cases where the medial wall of the middle ear and stapes are covered over by a firm matrix, dislocation of the stapes will be avoided.

8. In instances where the Eustachian tube orifice is sealed off by a firm matrix, this objective in radical surgery has already been accomplished.

WHEN THE MATRIX SHOULD BE REMOVED:

There are certain *anatomical* and *inflammatory factors* which make it advisable to remove the matrix.

A. Anatomical Factors.

1. A pneumatized mastoid. Most cases of chronically discharging ears with cholesteatoma are in sclerotic mastoids, but occasionally the mastoid will be partially pneumatic. Preoperative determination of this Xray is important since extensions of the cholesteatoma into pneumatic spaces can occur and will continue to expand if a thorough exenteration is not done. The matrix must be removed to expose the underlying cells.

2. Perilabyrinthine invaginations of the matrix into the bone should be searched for under magnification and should be removed. Such areas, however, can be dealt with without removing the entire matrix.

3. In revision mastoid operations, when one has not had a hand in the primary surgery, it may become necessary to remove the matrix to expose important landmarks and to determine the completeness or incompleteness of the previous surgery.

4. The matrix should be removed whenever there are retrofacial or petrous tip extensions.

5. If visible invaginations into bone in any area are excessive, the entire matrix in that area should be removed.

B. Inflammatory Factors.

1. Occasionally suppuration occurs between matrix and bone. This will manifest itself by the formation of granulation tissue between the matrix and the bone and by a pinkish color of the matrix. This elevated matrix should be removed and the bone searched for areas of necrosis.

2. There are cases in which islands of matrix are interspersed with islands of granulation tissue. This indicates bone infection and requires removal of the matrix for complete exposure to search for and clean out areas of bone necrosis.

3. The matrix should be removed when there is clinical or anatomical evidence of a suppurative labyrinthitis, petrositis, meningitis, subdural abscess, epidural abscess or lateral sinus thrombosis.

Another possible controversial issue is the suggestion that granulation tissue enveloping the ossicles be left alone if the removal is going to disturb the stapes or an intact ossicular chain, or if the removal destroys mucosa about the oval and round windows. I believe in removing all granulation tissue possible except in the situations just mentioned. The decision to leave granulation tissue is made mainly on an anatomical basis.[*] That granulation tissue formation is reversible is adequately proven by the good results in Cases 4, 7, 8 and 12, especially in 7 and 8, where the granulations left behind were considerable. It is possible that this ultra conservatism may require reoperation in some future case. Concerning this, my axiom to our residents-in-training is: "you can always take out more, but seldom can you put anything back." It is not a blot on one's escutcheon to have to reoperate a case if the initial surgery, done with an aim to preserve hearing, proves inadequate. *This inadequacy, however, must be a planned one and not because of inadequate technical knowledge or inadequate postoperative care.* I believe that the only complication to be concerned with in leaving granulations, as suggested, is this possibility of reoperation.

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[*] When the incus and the head of the malleus are removed in a case where the pars tensa of the tympanic membrane is intact and the middle ear is filled with granulation tissue, I sever the tendon of the tensor tympani and remove the midle ear granulation tissue except that which closely surrounds the stapes and the round window

It seems that the best hearing results obtained in cases where the incus and the head of the malleus have been removed, are in those that have a sealed tympanic space connected with a patent Eustachian tube and have a mobile stapes. Whether this tympanic space should be large or small has not as yet been determined. Martin[21] thinks that it is better for the space to be small. I am not sure that this is necessary but, frankly, I do not know. I have tried deliberately to keep the membrane from retracting toward the promontory, and Dr. Martin has tried deliberately to have it attached at some point to the promontory and to the lower medial attic wall. Suggestions for the accomplishment of both have been presented.

Apparently, when the incus and the head of the malleus are removed, a sealed tympanic air space is not an absolute requirement for hearing. In a few of Lathrop's[19] cases where he used a Lempert tympanomeatal flap with a perforation in the flap and the perforation did not close, these patients still had hearing. Just why is not clear. Possibly the tympanic membrane retracted against the stapes. Notwithstanding these successes, better results in the over-all picture will be obtained if the tympanic space is sealed.

The incidence of success, both as to hearing and dryness, will be low in any of these techniques if the after care is not meticulous and precise. The control of healing is as important as the operation.

There are other techniques, described elsewhere,[1,8,27] that are undoubtedly good and should not be ignored; however, those that have been presented are practical, not difficult, and have attained the sought-for end results.

It is apparent that we are in an era when the opportunity for salvaging an ear has never been greater. If our techniques and our thinking continue to develop "elasticity," it is certain that our future surgical accomplishments will be even more gratifying.

SUMMARY.

1. In surgery of the mastoid for a chronic inflammatory process, one should aim for a technique that will conserve or improve hearing.

2. This technique should be designed to produce a mobile stapes and an unscarred round-window-in-a-sealed tympanic air space in continuity with the Eustachian tube.

3. Techniques that have been successful in accomplishing this have been described. These include the management of various situations, with and without cholesteatoma, with and without preserving the cholesteatoma matrix.

4. In the ears that require surgery, the incidence of cholesteatoma is high. It is important to recognize that the matrix of the cholesteatoma can be converted to a useful structure, for when the matrix has sealed off the tympanic space in an ear with good hearing, leaving this matrix undisturbed will conserve or improve the hearing.

5. Various indications and reasons for preserving the matrix of a cholesteatoma, and indications for its removal, have been outlined.

6. The "key" to conservation of hearing in mastoid surgery, in addition to improved techniques, is to eradicate from our minds the obsession of intracranial complications, truly a bugbear of yesteryear, but only a shadow of a menace today.

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VASCULAR HEADACHES AND RELATED CONDITIONS.†*

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Pain, being one of the primary alarms of impending danger, impels most patients to seek medical advice quickly. Since sinusitis has been erroneously thought to be the cause of head pain, most of these patients with headache seek the advice of an otolaryngologist. The approach to this problem in the past has been primarily surgical. This was especially true if the patient was unfortunate enough to have an associated postnasal discharge or a vasomotor rhinitis which produced secondary changes in the mucosa of the nose or sinuses.

Unfortunate also, was the fact that the induced trauma from intranasal surgery gave temporary relief and led the surgeon to believe that this was the proper procedure; however, the relief in many cases was only transient. This trauma produced only temporary autonomic balance, probably due to the release of physiologic amounts of histamine into the organism. Soon, however, the unstable autonomic mechanism was again in dysfunction, since the basic cause had not been altered, and autonomic imbalance returned, expressing itself as head pain.[1]

The role of the autonomic nervous system and its relation to the cause of head and face pain has been recognized and given greater prominence in recent literature.[2,3,4]

We recognize that this autonomic or vegetative nervous system is responsible for all involuntary bodily function relating to blood vessels, glands and smooth muscle action. This involuntary nervous system is constantly at work attempting to equate the organism with its everchanging environment.

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The center of activity of this system is located in the hypothalamus. Here the combined activities of the autonomic nervous system are integrated. Reflexes from body surface receptors, reflexes from the Vth, VIIth, IXth and Xth cranial nerves, and the first three cervical nerves; and impulses from psychomotor levels are relayed through this central communicating area to peripheral organs located in muscular, secretory and vascular endings. When this integration is inadequate or faulty, disorganized and imbalanced function results.

The nature of one's autonomic nervous system represents the composite of those of his forbears. It may be that this system, functioning well for them, cannot continue to function for us under the constant barrage of modern-day stress and strain. This disorganized response may also be caused by more tangible factors which Hilger[4] classifies, in addition to the emotional, as physical, inhalant and ingestant. This inability to cope with one's environment is known as autonomic dysfunction. The effect of this dysfunction on the carotid arterial tree is a major cause of vascular headache.

HISTORY.

An adequate, detailed and complete history of the patient must be taken, keeping in mind that the patient is a whole organism and not a group of semi-isolated organs functioning independently of one another. The following points are stressed:

1. *Onset*—Age and circumstances of the first attack.

2. *Known Contributing Causes*—An exhaustive family and personal medical history is taken, stressing emotional factors, tobacco, allergies, fatigue, infection and menstruation.

3. *Prodromata*—Duration and symptoms during premonitory state.

4. *Location of Head Pain.*

5. *Quality*—Is it constant, boring or burning? Is it an abrupt or gradual onsent?

6. *Timing*—The frequency and duration of attacks. Result of previous medication on this timing.

7. *Effect on Headache*—Of position or movement of the head; of the effect of heat, cold, noise, light, eating and drinking.

8. *Associated Manifestations During the Headache*—On the eye, nose, throat, ear, neck. Is edema present? Frequency of urination, etc.

9. *Termination of Attack*—After sleeping, after activity. Is there sweating, local soreness, etc.

Following the history, a complete physical examination is made including, where necessary, laboratory, X-ray, medical, ocular, dental, orthopedic and neurological consultations.

MECHANISM OF VASCULAR PAIN.

There are several theories relative to the mechanism of pain of vascular headache. Hilger[1,4,5,6] pictures all vascular headaches as being fundamentally one and the same, the only difference being in the initiating cause, in location and in degree of vascular imbalance. According to him, the cause is vasospasm at the arteriolar level. This vasospasm is caused by some poorly integrated autonomic stimulus. Ischemia develops in the distal arteriolar-capillary-venular portion of this end-of-the-line unit, causing damage to the capillary endothelium with resultant dilatation, leakage and extravascular transudate.

If the involved arterioles are terminal, they erect an effective dam, causing proximal, passive vasodilatation due to the increased amount of blood pounding up against the constricted arteriole. If the arterioles are located within the walls of the parent artery, in the vaso-vasorum, the ischemia and resultant transudate cause a flaccidity of the muscular coat, with resultant vasodilatation. Either of these causes of dilatation stretches the peri-arterial sensory nerve plexus, with resultant pain.

Other theories, too, attempt to explain this pain mechanism by changes in the peripheral organs, but it should be remembered that evidence supports the theory that pain can arise from abnormal activity of the central nervous system without peripheral changes, and a good example of this is phantom limb pain. Despite the fact that the pain of migraine has been shown to be in proportion to the degree of dilatation and pulsation of the external carotid artery,[7] it has also been shown that cranial vasodilatation, either generalized or localized, can be produced by various chemical agents *without pain;* furthermore, it may often be noted that, following an attack of migraine, the vessels are still edematous and dilated long after the pain has subsided. Friedman[8] gives a more complete description and references to the above mentioned theories.

It may be that the final accepted theory of the mechanism of pain will be one embracing a combination of all of these theories, just as it is generally recognized that a headache can be caused by a combination of factors—fatigue plus allergy, autonomic imbalance plus bodily depletion, etc. I think that such a theory is possible when one considers that ergotamine and its derivatives, which relieve a majority of attacks of headache, have not only a constrictor action, but also a central and sympatholytic effect as well. In other words, vasodilation is not the final answer, or else other vasoconstrictors would be equally effective. Central overstimulation is not the only explanation, because drugs inhibiting the central nervous system alone are seldom of help. Sympathetic hyperaction is not the complete answer, because all sympatholytic drugs are not efficacious. When one considers the pharmacology of the drugs which give relief of pain in migraine, this theory seems plausible.

CLASSIFICATION OF HEADACHES AND THERAPY.

As stated before, the initiating cause or causes of vascular headache are legion. Hilger[5,6] has stated that emotional factors influence peripheral autonomic expression through direct associational connection between frontal brain centers (emotional) and the hypothalamus, while physical factors of humidity, barometric pressure, temperature, light, etc., involve reflex mechanisms between body surface receptors and peripheral autonomic expressors; also that inhalants and ingestants create environmental stress which on re-exposure exhibit, through some autonomically inherited capacity, an antigen-antibody conflict. This is recognized as allergy in the narrow sense of the word.

That vascular headache can be secondary to irritation of cranial nerves has been reported by several authors. Douglas[9] reported facial pain secondary to elongated styloid process, and Costen[10] has reported numerous cases of facial pain secondary to tempero-mandibular joint dysfunction. Hunter and Mayfield[11] reported eleven cases of vascular headache secondary to irritation of the second cervical sensory root. These eleven cases were treated by section of this root; however, immediate relief was obtained only in those cases which had had a definite history of trauma to this nerve.

It is agreed that allergic reactions, states of autonomic dysfunction and endocrine imbalance can produce identical clinical symptoms, and that the common denominator is excessive and abnormal extravascular fluid in the end-organ tissue. It is agreed also that it is not enough to treat vascular pain in a standard predetermined stereotyped manner. We must recognize, as did Hunter,[11] that successful treatment is obtained only in eliminating the offending cause. Psychotherapy will hardly help a headache originating from an irritated second cervical root, and section of this root will be of questionable aid in headaches of allergic origin; but we do know that there is a relationship between emotional factors and the migraine type headache; that there is a correlation between physical changes of weather and the myalgia type headache.

This point supports our opinion, we believe, that a broad classification of headache types is indicated on a practical clinical and therapeutic basis. It is a guidepost for the physician, helping him relieve the patient's symptomatic complaints on his first visit; it also gives the physician thinking time. An injection of nicotinic acid for the myalgic patient, or an injection of DHE-45 for the migrainous patient sends these people away from the office happily relieved of their headaches, while measures can be taken prophylactically, to prevent their second attacks. Thus we add a little psychotherapy to our pharmacotherapy.

If on the other hand we delay our therapy too long while we attempt to get far enough back from the patient, so as not to be accused of narrow specialism, we may lose forever our chance to help him; therefore, a basic working classification of classical headache entities is indicated. We feel that this is important from the standpoint of basic clinical diagnosis and from symptomatic and prophylactic therapy. (Many authors have presented their classifications of headaches, and to prevent needless repetition, the physician is referred to them.[7,8])

A few of the more commonly encountered classical head pains will be defined here, and the therapy, both symptomatic and prophylactic, will be discussed.

1. *Migraine:* Migraine is a vascular, unilateral, severe, recurring headache with a variable period of relief between attacks, and occurring against a background of relatively good health. There is usually a family history present. Migraine usually starts with an aura characterized by visual and emotional disturbances. This aura is followed by a throbbing headache, and frequently nausea. As the attack progresses, the pain loses its throbbing quality and becomes more constant. It is more common in women than in men. The theory of the mechanism of migraine has been established from these progressive symptoms; that is, the aura is associated with the pre-constriction phase, the throbbing headache with vaso-dilatation, and the constant headache with edema. An attack of migraine may last from a few hours to many days.

An acute attack of migraine and migraine-like headaches may be absorbed or controlled by various drugs, principally the vasoconstrictors. The treatment of choice is the early and adequate administration of Cafergot, gynergen, or DHE-45 Cafergot is now available in suppository form for those patients whose nausea precludes oral medication; valoctin may also be considered. Prophylactic measures consist primarily of psychotherapy. Migraine patients are usually perfectionistic, rigid, over-conscientious, meticulous, hostile and generally dissatisfied with their station in life. They must be taught new attitudes to reduce tension and the proper balance of work, play and rest must be maintained. Drug therapy is indicated as an aid to psychotherapy if the drug is capable of inhibiting autonomic overflow, and bellergal,[3] the autonomic inhibitor of choice, has been found to be effective for this purpose. Nicotinic acid and histamine desensitization may also be considered.

2. *Histaminic Cephalgia:* According to Horton,[12] this headache "is characterized by unilateral headache, which usually begins in the later decades of life, is of short duration, as it generally lasts less than an hour, commences and often terminates suddenly, tends to awaken the patient at night one or two hours after he has gone to sleep, and is frequently eased by the patient's sitting up or standing erect. It is associated with profuse watering and congestion of the eye, rhinorrhea or stuffiness of the nostril, increased surface temperature, and often swelling of the temporal vessels of the involved side of the head. Pain is the outstanding complaint. It is constant, excruciating, burning and boring; it involves the eye, the temple, the neck and often the face." It is more common in men.

The acute attack of histaminic cephalgia may be controlled in the same manner as migraine with Cafergot and gynergen, or DHE-45. Prophylactic therapy consists of psychotherapy, bellergal and desensitization with histamine diphosphate. The optimum dosage technique of administering histamine diphosphate, as described by Hansel,[13] has been found to be most effective, especially for office practice. This technique is carried out as follows: the beginning dosage is 0.1 cc. of No. 6 dilution (1:1,000,000) or No. 7 dilution (1:10,000,000), given intradermally daily to twice weekly. If this dosage is too small no effect will be noted, and the dosage should be increased to stronger dilutions. If and when the dosage is too large, an attack of headache will be precipitated. When the optimum dose is reached the patient will be symptom-free. The dosage interval is then increased gradually to one injection every 14 to 15 days. This is continued for about three months and then discontinued.

Temporary prophylactic use of Cafergot suppositories during frequent bouts of these headaches is also effective. These are inserted at bedtime. (Many times one-half of a suppository is sufficient).

3. Tension: This type of headache usually occurs at the height of, or during the let-down period of emotional conflicts or stress situations. It may be conscious or unconscious. Gastrointestinal disturbances are usually not present. The quality of pain is of a constant, boring nature, in contrast to the throbbing quality of the migraine headache. It is always associated with pain in the occipital region. This is thought to be due to the fact that there is a concomitant spasm of the skeletal muscles of the head and neck *and* dilatation of the cranial vessels.

Tension headaches have no prodromata, are usually bilateral, occipital or frontal, and may be accompanied by a variety of associated signs, including anxiety, nausea and vomiting. Frequency and duration are variable. There is no aura and no family history is present. It is more common in women than in men. Combined analgesics and barbiturates (APC and phenobarbital) are effective for relieving the acute attack of tension headache. Prophylactic therapy is essentially the same as described for migraine.

√ *4. Myalgia:* As vasospastic head or neck ache, occurring usually after the second decade of life. It is unilateral and involves the occiput and (or) the pharynx. The pain is of the deep type, rising slowly to a crisis and slowly subsiding. It occurs after severe illness or exposure to cold, dampness or changes in weather, and for this reason is often erroneously diagnosed as an infection. The process is thought to be one of physical sensitivity.[15] Any or all of the neck, throat, and pharyngeal muscles may be involved. It is caused by arteriolar spasm in these muscles. (One of the most common examples of this is myalgia of the superior constrictor of the pharynx—the patient has an intense sore throat without physical findings).

Intramuscular or intravenous nicotinic acid is the treatment of choice for symptomatic management of myalgia. Protection from exposure to drafts, air-conditioned rooms, and abrupt weather changes will modify the frequency of attacks of myalgia. Prophylactic use of histamine and oral nicotinic acid is also of value. (Dosage of I. V. nicotinic acid usually begins at 15-25 mgm. and increased to tolerance, one to two times weekly).

5. *Carotid:* There are a number of other related head pains which are vascular in nature and which may be called "carotid," in that they involve other branches of the external carotid artery. These pains are located in the face, head, neck and teeth, but differ from the headaches already described in quality, duration and severity of the pain. As in the case of the other vascular headaches, they were formerly diagnosed as, or caused by sinusitis, otitis media, infection.[1,14] Many of Sluder's lower-half headaches must have been of this nature.

The symptomatic and prophylactic treatment of carotid pain taxes the ingenuity of the physician and may involve any one, or a combination of the previously outlined therapy. Histamine desensitization, nicotinic acid (I.V. and oral) and bellergal give the most consistent results in a majority of the cases.

6. *The Capillary Loop:* The physiology of the affected capillary loop may be restored to normal by administration of large amounts of vitamins B and C, thyroid, and the electrolytes including potassium and calcium. Where there appears to be a gonadal or endocrine etiology the use of hormones and steroids is indicated.

A recommended treatment for the edema phase of any vaso-dilating headache is the use of neo-calglucon 20 per cent intravenously. The total amount of 10 cc. is injected as follows: 3.0 cc. is injected, rapidly followed by a pause until flushing fades; this is repeated, followed by a second pause and the final 4.0 cc. is then injected.

For obvious reasons, drug therapy has its limitations.

When a patient's personality shows him to be a highly disciplined, perfectionistic, tense person, he must be shown how to re-orient himself to other people and to other ways of life. The doctor who takes time to teach his patient new attitudes to reduce tension will enable him to meet his responsibilities with less strain and may reduce the number and severity of attacks, even though their origin is probably never enentirely psychic.

Dr. English[10] suggests the following principles which will be helpful to the patient if he can be taught to make use of them:

1. This is not a perfect world. Families and friends have many foibles. Perfection is rarely attained, so be satisfied with less.

2. Tolerance makes understanding the other fellow easier. It sets an attainable standard.

3. Do not be a slave to the clock. Work at your own pace; do as much as you can. Trying to meet too many deadlines only creates tension.

4. You cannot please everybody, so stop trying. Popularity comes by giving your friends and family a chance to love you for yourself, not for your best performance.

5. Be efficient, yes, but not to the extent that perfection becomes a burden.

6. Speak up if you want to. You cannot please everybody; honesty and directness break down barriers and make friendships easier.

7. Approve of yourself. You are as good as the next fellow. If you are given a compliment, take it and make use of it.

8. Stop being so critical of your negative feelings. Everyone is ambivalent at times, so do not worry so much about loving and hating.

9. Stop feeling guilty. We are all human beings and we all make errors. Give a little and you will get a lot, maybe even a reduction of that pain in your head.

The success of the prophylactic treatment of headache depends, in large part, on the able handling of the symptomatic phase; but, regardless of the type of headache, consideration must be given to general health, metabolic changes, nutrition, endocrine and autonomic imbalance, allergy, environment, and the protoplasm of the patient. In short, the patient must be treated as a whole organism.

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THE FENESTRATION OPERATION IN THE IDEAL CANDIDATE.*†

215 Cases Selected From 482 Operations.

J. BROWN FARRIOR, M.D.,

Tampa, Fla.

In the ideal candidate with the ideal fenestration operation the patient should always receive a maximum and permanent restoration of hearing. With good present-day technique with the Lempert fenestration operation these patients almost always receive a good result, and the restored hearing almost always lasts. Actual closure of the fenestra is extremely rare, but there remain many variables which prevent consistent uniform perfect results.

Of these variables the greatest is in the quality of the surgery. Secondly, is the patient's response to the surgical trauma. In selecting the ideal candidates, it is assumed that the errors of diagnosis have been eliminated. To determine the base level for evaluation of our current surgical technique, results in these ideal candidates are analyzed.

For purposes of this study the 215 ideal candidates have been selected from the second, third and fourth hundred fenestration operations in a total series of 482 fenestrations. Their average age is 35.1 years.

DEFINITION.

The ideal candidate for the fenestration operation is the otosclerotic patient with a flat or ascending air conduction audiogram, with a 40 to 60 decibel hearing loss for the speech frequencies, and normal bone conduction.

The recording of normal bone conduction will vary with

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* Read at the meeting of the Southern Section of The American Laryngological, Rhinological and Otological Society, Inc., Louisville, Ky., January 16, 1954.

† From the Department of Otolaryngology, Tampa Municipal Hospital, Tampa, Florida.

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the audiometer and with the testing conditions. The present day audiometer is calibrated so low that perfect conditions are required to obtain a zero reading. Under average office conditions, a bone conduction reading at the 15, 20, or 25 decibel level is to be regarded as normal.

The normal base level of 20 db having been determined for the testing conditions, the otosclerotic patient will demonstrate an additional 5 and 10 db lower recording at 1000 and 2000, as described by Carhart. In the otosclerotic patient this Carhart notch is normal bone conduction.

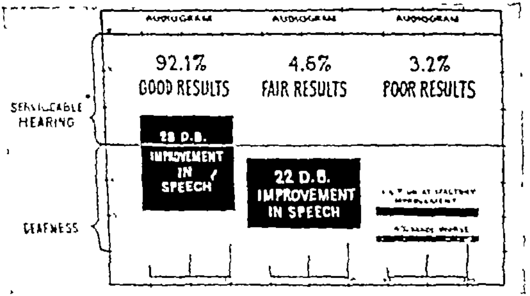

Fig. 1. Results in Ideal Candidates. In 215 ideal candidates, 96.7 per cent showed a worthwhile clinical result. More detailed testing and analysis shows that there is plenty of room for improvement in technique. (From Archives of Otolaryngology, July, 1953).

PRIMARY RESULTS.

The standard method of reporting results of fenestration surgery is based upon the average decibel gain and level for the speech frequencies. By this basic method of reporting, 92.1 per cent of the 215 ideal candidates had good results with hearing well above the critical 30 decibel level, 4.6 per cent received a fair result, with hearing improvement to near the 30 decibel level, 1.8 per cent received a partial but unsatisfactory improvement, and 1.4 per cent were made worse. The decibel level and gain are shown in Fig. 1.

Combining the good and fair results, 95.7 per cent of the ideal candidates received worthwhile hearing improvement by this basic method of reporting.

LASTING RESULTS.

With current fenestration techniques, the lasting restoration of hearing is an accepted fact in the overwhelming majority of cases. Bony closure is rare. The standard methods of reporting are the fallible questionnaire and the factual audiogram.

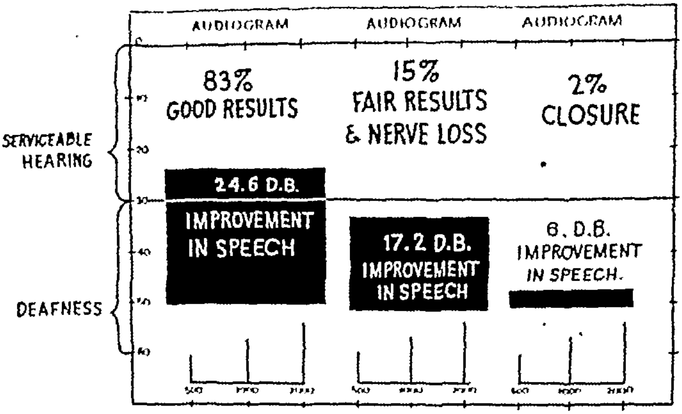

Fig. 2. Lasting Results. Two- to five-year post-operative audiograms on 100 good primary results showed that 83 per cent retained hearing at or above the critical 30 db. level; 15 per cent retained a partial and satisfactory hearing improvement; and actual bony closure in 2 per cent.

With the fallible questionnaire with 97 per cent replies in the 215 ideal candidates there has been 2.3 per cent closure. By the fallible standard of measure of the 96.4 per cent which obtained improvement, 97.7 per cent have maintained a worthwhile hearing improvement.

It is almost impossible to obtain the factual two-year postoperative audiogram on consecutive cases, even though we do have nearly three thousand postoperative audiograms.

Reviewing these, in three- to five-year-old cases (Nos. 100-250), there are two- to five-year-old postoperative audiograms on 100 patients previously reported as good results. The final audiogram is three, four, or five years postoperative in 78 per cent. Analysis of these 100 mature audiograms, including all closures on previously classified good results, reveals that 2 per cent are closures, 15 per cent have changed from good to fair results, and 85 per cent have remained good results (see Fig. 2).

The 15 per cent long-term fair results have maintained an average gain of 17.2 decibels at the 33.5 decibel level. Three of these showed a comparable nerve loss in the opposite ear. The 83 per cent long-term good results have maintained an average gain of 24.6 to 24.7 decibel level.

The 98 patients (83 per cent and 15 per cent with good and fair results) are pleased with their long-term hearing improvement, and give a favorable reply to the questionnaire; therefore, it is reasonable to assume that in favorable replies to the questionnaire, 15 per cent have maintained a partial hearing improvement, which is just below the critical 30 decibel level, and that the remainder are good results. Further, by any standard of measure, it is reasonable to assume that the incidence of closure is less than 5 per cent.

IMPERFECTIONS.

If we are to perfect our already refined fenestration technique we must have more delicate methods of recording and reporting results, both immediate and lasting.

Already available in everyone's records is the effect of the fenestration operation upon the high tones. A loss of hearing for these high tones is considered to be evidence of minor but irreversible serous labyrinthitis. As shown in Table I, in nearly 500 fenestrations, I have improved only 5 per cent in ability to preserve hearing for the delicate high tones. The high tones were maintained in 76.7 per cent, 76.4 per cent, 77.8 per cent and 81.9 per cent of the first, second, third, and fourth hundred fenestrations.

It is interesting to note that the ideal candidates comprised 51 per cent of the first hundred, and 70, 73 and 72 per cent of the second, third and fourth hundred fenestration operations.

The type, degree and duration (see Fig. 3) of postoperative labyrinthitis should be recorded to determine the immediate effect on the inner ear of supposed refinements in technique. We have classified postoperative labyrinthitis into primary surgical labyrinthitis, secondary serous labyrinthitis, and tertiary or degenerative cicatricial.

TABLE I. HIGH TONES MAINTAINED

Cases	No. Ideal Cases	Per Cent H. T. Maintained
0 - 100	51	76.7%
100 - 200	70	76.4%
200 - 300	73	77.8%
300 - 400	72	81.9%

Table 1: The loss of high tones is minor but irreversible evidence of serous labyrinthitis. In 482 fenestrations the author has shown only 6 per cent improvement in the ability to maintain high tone hearing, indicating existing imperfections in fenestration technique.

The primary surgical labyrinthitis is the immediate effect of surgical injury or hemorrhage, and is demonstrable on the first postoperative day. The secondary serous labyrinthitis is the result of the inflammatory response, and reaches its peak on the fifth to tenth postoperative day. Both of these may be reversible. The tertiary degenerative or cicatricial

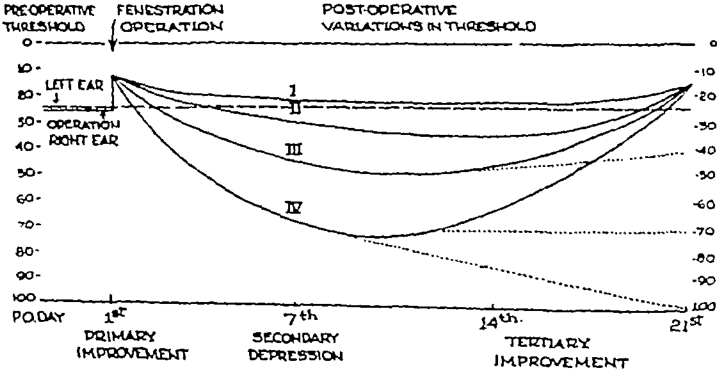

Fig. 3. The post-operative course of bone conduction threshold may be divided into three phases; primary improvement, secondary depression, and tertiary improvement. Primary improvement may be altered by surgical trauma, operative hemorrhage, or round window fixation. The secondary depression varies with the degree of serous labyrinthitis. Tertiary improvement usually occurs, but there may be permanent sequellae as loss of high tones of nerve deafness. Post-operative labyrinthitis is therefore classified as primary surgical, secondary serous and tertiary degenerative.

labyrinthitis is the irreversible end result. The loss of high tones is the mildest form, and nerve deafness the severe form. A late nerve loss may be the end result of surgical trauma, or a process of nature; the latter is assumed if a comparable process is exhibited in the unoperated ear.

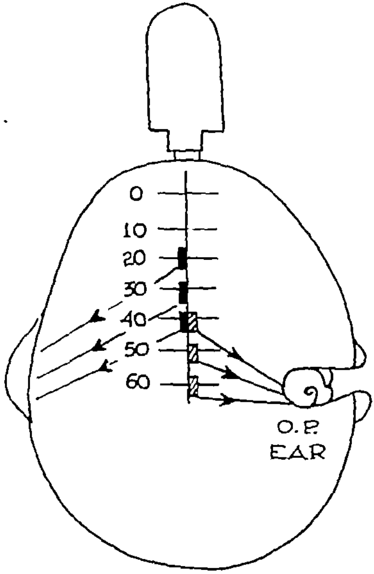

Fig. 4 The phenomenon of recruitment may be demonstrated by bone conduction with the "Calibrated Weber" test. In a selected series 45 per cent showed this undesirable recruitment, by a shift of lateralization from the unoperated ear to the operated ear with increasing intensity of sound of the centrally placed bone conduction occillator.

To measure the type, degree and duration of postoperative labyrinthitis, we have adapted the Weber test to the bone conduction oscillator of the audiometer. With the Calibrated Weber Test[1] it has been possible to demonstrate variations in threshold and, with increasing intensity, variations in the lateralization of sound in the recently fenestrated ear. The threshold of bone conduction was found to be improved by improved mobility of the perilymph, or depressed by trauma, hemorrhage or serous labyrinthitis. In 40 of these ideal cases, the calibrated Weber was found to lateralize to the operated ear from two different causes: *1*. The desirable physiological improvement in threshold; or *2*. the undesirable pathological recruitment (see Fig. 4). In these 40 cases only 75 per cent showed the desirable primary improvement in threshold on the first postoperative day, indicating that in 25 per cent there was some degree of primary surgical labyrinthitis, as shown in Fig. 3.

The secondary serous labyrinthitis (see Fig. 3, curves I, II, III, IV) was found to be minimal (I) in 17.5 per cent, mild (II) in 35 per cent, moderate (III) in 45 per cent, and severe (IV) in 2.5. per cent.

Of the three cases which comprise the 1.4 per cent of cases which were made worse, two cases were technical errors, and one was an error of judgment. In one case the membranous labyrinth was injured with the pick, in one case the membranous labyrinth was injured by the burr. In the error of judgment I thought I had cured the preoperative external otitis but the patient developed an infection and lost his hearing.

On the brighter side of this analysis, 96.7 per cent of these ideal candidates showed a worthwhile clinical and practical hearing improvement, which is gratifying to the patient and surgeon. Idealistically, in this 96.7 per cent there is plenty of room for improvement. About 22 per cent showed loss of hearing for the high tones, 4.6 showed only partial improvement, and with the Calibrated Weber in 40 cases, 25 per cent showed primary surgical labyrinthitis, about 50 per cent showed undesirable degrees of secondary serous labyrinthitis with recruitment.

Fortunately most of these processes are reversible, and the patient obtains a desirable clinical result; yet none of these undesirable processes should occur with the ideal fenestration technique.

FENESTRATION TECHNIQUE.

The fenestration techniques employed today are highly refined, and make the fenestration operation one of the most gratifying of all surgical procedures. There are many additional refinements which are under investigation today. I will mention a few:

1. Lempert's cupola technique should produce minimal primary surgical labyrinthitis. Invagination of the flap compensates for the fractured edges of the fenestra to secure intimate approximation between the endosteum and the under surface of the flap, encouraging healing by primary intention and permanent patency of the fenestra.

2. Shambaugh is investigating the possibility of covering the fenestra with the primary skin graft, reasoning that the inert skin graft will reduce the degree of postoperative serous labyrinthitis.

3. The narrow pedicle flap vs. skin graft over the fenestra. To reduce the postoperative inflammatory response within the flap we have, since 1947, utilized the narrow pedicle flap attached to the drum only at the malleolar folds, reasoning that this would produce an almost inert flap with minimal postoperative vaso-dilatation and exudation into the inner ear. Recently we have used such a skin graft in three instances. In two there was good lateralization to the operated ear, good improvement in threshold and good retention of high tone hearing; in one there was partial depression of base conduction, shift of lateralization and loss of high tones, but a good gain for speech. We plan to continue using the skin graft to cover the fenestra in those cases where there is a significant imperfection in the flap.

4. We have used Lamp's isothermal isotonic irrigation technique with little apparent change in the postoperative course, as determined by the Calibrated Weber Test. A detailed analysis is now in process.

5. Hollow ground margins. To maintain permanent patency of the fenestra, all operations strive toward obtaining healing by primary intention between the endosteal margins of the fenestra and the under surface of the flap. In our technique we concentrically hone the margins of the fenestra with

the finishing and smooth burr, to obtain sharp elevated hollow ground margins to obtain intimate approximation between the endosteal margins and the under surface of the flap (see Fig. 5).

COMPLICATIONS.

In the entire series of 482 cases, there have been no serious complications and no deaths. A detailed description of the nuisance factors of fenestration surgery has been presented in the January *Archives of Otolaryngology.*[2]

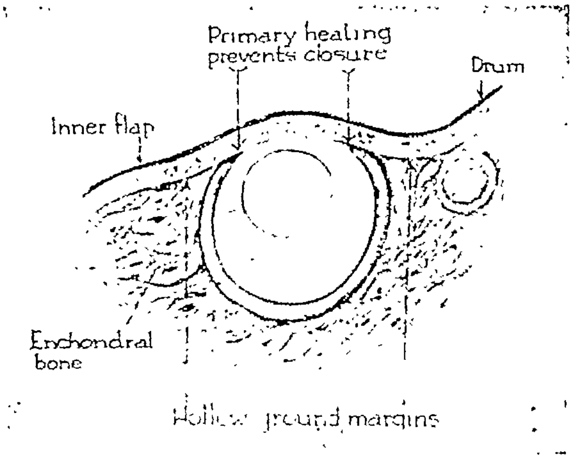

Fig. 5. Hollow Ground Margins. A section of the fenestra shows the concentrically honed sharp hollow-ground margins which elevate the endosteum to intimate approximation with the under-surface of the flap, insuring healing of the margins by primary intention and therefore, permanent patency of the fenestra. (From Archives of Otolaryngology, July, 1953).

CONCLUSIONS.

The present standard methods of reporting results do not reveal the imperfections of fenestration surgery. The present day fenestration techniques are highly refined, and will almost always obtain and maintain a good clinical result in ideal candidates; yet there remains plenty of room for improvement.

If we are to determine the effect of further refinement of fenestration techniques, more detailed methods of reporting are necessary. The effect of the high tones and the type, degree and duration of labyrinthitis should be recorded.

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509 Bay-Shore.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1 and continue for a period of ten months. Registrations must be completed before August 15. Detailed information and application forms may be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minnesota.

PROGRAM OF MEETING OF THE WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

To Be Held

MAY 27th TO 29th, AT THE GREENBRIER HOTEL,
CHARLESTON, WEST VIRGINIA.

Thursday, May 27th

8:30 P.M.—Business Meeting

Friday, May 28th

9:00 A.M.—Call to order by President, J. K. Stewart, M.D.

9:20 A.M.—"End Results of Post-Enucleation Orbital Implants." By Roscoe Kennedy, M.D., Cleveland, Ohio

10:30 A.M.—"Rhinoplastic Approach to Treatment of Recent Nasal Fractures."—F. C. Reel, M.D.

11:15 A.M.—"Retrolental Fibroplasia."—C. F. Breisacher, M.D., and M. J. Lilly, M.D.

1:00 P.M.—Lunch

2:00 P.M.—Golf and Tennis

7:00 P.M.—Social Hour followed by banquet, dancing, etc.

Saturday, May 29th

9:30 A.M.—"Differential Diagnosis of Deafness in Early Childhood"—H. E. McHugh, M.D., Montreal, Canada

10:40 A.M.—"The Treatment of Ulcerative Keratitis with Vitamin B-Complex"—Joseph S. Maxwell, M.D.

11:10 A.M.—"Vocal Cord Paralysis—Diagnosis and Office Management"—William K. Marple, M.D.

11:40 A.M.—Progress Report on West Virginia School for the Deaf and Blind—Mr. Hugo F. Schunhoff, Superintendent.

12:00 Noon—Business Session—Election of Officers.

The business meeting scheduled for Thursday evening will be important, and essentially the only one, for that scheduled for Saturday noon will be very brief, probably has been given over just to election of officers. An enlarged entertainment committee has been at work on that part of the meeting and while no rigid program of ladies' events has been scheduled there will be something for everyone to do.

NINETEENTH INTERNATIONAL CONGRESS OF NEURO-OPHTHALMOLOGY AND NEURO-OTOLOGY.

June 11 to June 17, 1954, San Paulo, Brazil.

OFFICIAL PROGRAM.

The city of Sao Paulo is commemorating the fourth centenary with a series of festivities and activities of widespread interest. Of particular interest are various artistic, cultural, literary and scientific congresses. Among these is the Nineteenth International Congress of Neuro-Ophthalmology and Neuro-Otology. The date of this congress falls at a time when the Sao Paulo climate is particularly pleasant. In addition to the scientific program, there will be many activities of social and artistic interest. Many of these will be exclusively planned for the entertainment of the ladies. The official program of this congress is as follows:

A. Vitamin deficiency and metabolic disturbances in neuro-otology and neuro-ophthalmology.

1. Introduction to the study of Metabolic disturbances. Prof. A. Kestenbaum, Neuro-ophthalmologist, New York.
2. Neuro-otologic and Neuro-ophthalmologic disturbances of lipoid metabolism. Prof. Diogo Furtado, Neurologist, Lisbon, and Prof. A. Francheshetti, Ophthalmologist, Geneva, Switzerland.
3. Neurologic and Ophthalmologic manifestations of abnormal carbohydrate metabolism. Prof. H. K. Muller, Ophthalmologist, Bonn, Germany, and Prof. H. Leicher, Otorhinolaryngologist, Mainz, Germany.
4. Neurologic and Ophthalmologic manifestations of abnormal protein and mineral metabolism. Dr. Flaminio Vidal, Ophthalmologist, Buenos Aires, Argentina.
5. Neurologic and Ophthalmologic manifestations of vitamin deficiency (A, D & E). Prof. Franklin de Moura Campos, Physiologist, Sao Paulo Brazil. Prof. Mario Gozzano, Neurologist, Rome, Italy. Prof. P. Desvignes, Ophthalmologist, Paris, France.

B. Physiopathology of the facial nerve.

1. Paralysis of the Facial Nerve

Pathology—Prof. Raymond Garcin, Neurologist, Paris, France.

Therapy—Prof. Pereyra Kafer, Neurologist, Buenos Aires, Argentina.

2. Facial Nerve Spasm

Pathology—Prof. Karsten Kettel, Otorhinolaryngologist, Hillerod, Denmark.

Therapy—Dr. Aloisio Mattos Pimenta, Neuro-surgeon, San Paulo, Brazil.

In addition to meetings limited to the official program, there will be other sessions at which free papers will be presented. For information write: Ophthalmologic Clinic. Hospital das Clinicas, Sao Paulo, Brazil.

IVe CONGRES DE L'ASSOCIATION INTERNATIONALE POUR L'ETUDE DES BRONCHES.

Genève, 5-6 juin 1954

Cet important congres qui se tiendra a Geneve, sous la presidence du Professeur Montandon, directeur de la Clinique universitaire d'O.R.L., de Geneve, au printemps prochain, a mis a l'ordre du jour de ses seances les questions suivantes (chaque question etant traitee par 3 rapporteurs) :

I. La vascularisation sanguine des bronches: *Rapporteurs:* Prof. Bruni (Milan), Prof. von Hayek (Vienne) et Prof. agr. Latarjet (Lyon).

II. La place nosologique des "adenomes bronchiques de Jackson"; Rapporteurs deja annonces: Prof. von Albertini (Zurich) ; Prof. gr. Dr. Galy (Lyon).

III. Les indications et les resultats des traitements chirurgicaux des bronchiectasies: *Rapporteur* deja annonce: Prof. Brunner (Zurich).

Pour tous renseignements, s'adresser au Prof. A. Montandon, *Clinique*, universitaire d'O.R.L., Hospital cantonal, Geneve.

DEUTSCHE GESELLSCHAFT DER HALS-NASEN-OHRENARZTE.

The 25th Annual Meeting of the Deutche Gesellschaft der Hals-Nasen-Ohrenarzte will be held June 2 and 5, 1954, at Marburg-Lahn. For further information, address Prof N. Mittermeier, Marburg-Lahn, Germany.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

May 1, 1954.

Acousticon Models A-17, A-180 and A-185.

Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 1, New York.

Auditone Models 11 and 15.

Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67 and 70.

Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac Model M; Mono-Pac Model "Lyric"; Mono-Pac Model "Rhapsody."

Manufacturer: Beltone Hearing Aid Co., 2900 West 36th St., Chicago 32, Ill.

Cleartone Model 500; Model 700; Cleartone Regency Model.

Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Model D-1; Dahlberg Junior Model D-2; Dahlberg Model D-3 Tru-Sonic; Dahlberg Model D-4 Tru-Sonic.

Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Fortiphone Models 19-LR; 20A; 21-C and 22.

Manufacturer: Fortiphone Limited, Fortiphone House, 247 Regent St., London W. 1, England.
Distributor: Anton Heilman, 75 Madison Ave., New York 16, N. Y.

Gem Hearing Aid Model V-60.

Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.
Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico Model J; Maico Top Secret Model L; Maico Maxitone.

Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis, Minn.

Micronic Model 303; Micronic Model "Mercury"; Micronic Star Model.

Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Classic Model T9; Microtone Model T10; Microtone Model T612.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 106 So. 7th St., Philadelphia 6, Pa.

Normatone Model C and Model D-53.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.

Distributor: Normatone Hearing Aid Co., 22 East 7th St., St. Paul (1), Minn.

Otarion Models B-15 and B-30; Otarion Models F-1; and F-3; Otarion Model G-3; Otarion Model H-1; Custom "5."

Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40, Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny-Myte); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Model 62 Starlet; Model 72; Model 82 (Zephyr).

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Distributor: Radioear Corp., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Silvertone Model H-16, J-92; Silvertone Model P-15.

Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.

Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966; Sonotone Model 977; Sonotone Model 988.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Televox Model E.

Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 953; Telex Model 1700.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.

Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster; Model Cameo.

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Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2.

Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal; Zenith "Regent."

Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices have vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TRANSISTOR HEARING AIDS ACCEPTED.

Acousticon Model A300; 1 transistor, 2 tubes. Model A-310; 1 transistor, 2 tubes. Model A-330; 3 transistors.

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Audivox, Model 71; 3 transistors.

Manufacturer: Audivox, Inc., 123 Worcester St., Boston 18, Mass.

Beltone Concerto Model; 3 transistors.

Manufacturer: Beltone Hearing Aid Co., 2900 W. 36th St., Chicago 32. Illinois.

Maico Transist-Ear, Model O; 3 transistors.

Manufacturer: The Maico Company, Inc., 21 N. 3rd St., Minneapolis 1, Minnesota.

Micronic "All American" Hearing Aid; 3 transistors.

Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Model T1 (Red Dot); 3 transistors. Microtone Model T1 (Yellow Dot); 3 transistors. Microtone Model T31 (Micro-Mite); 1 transistor and 2 tubes.

Manufacturer: The Microtone Corporation, Ford Parkway on the Mississippi, St. Paul 1, Minn.

Otarion Model C-15; 1 transistor, 2 tubes. Otarian Model F-22; 1 transistor, 2 tubes.

Manufacturer: Otarion, Inc., 4757 N. Ravenswood Ave., Chicago 40, Ill.

Radioear Model 820; 3 transistors.

Manufacturer: E. A. Myers & Sons, Inc., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Silvertone Model H-25; 3 transistors.

Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.
Distributor: Sears, Roebuck & Co., 925 South Homan Ave., Chicago 7, Illinois.

Sonotone Model 1010; 1 transistor, 2 tubes.

Manufacturer: Sonotone Corporation, Elmsford, N. Y.

Telex Model 954; 1 transistor, 2 tubes.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Unex Model TR-3D; 3 transistors.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Zenith Model Royal-T; 3 transistors. Zenith Model Super Royal-T; 3 transistors.

Manufacturer: Zenith Radio Corp., 5801 W. Dickens Ave., Chicago 39, Illinois.

SEMI PORTABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).

Manufacturer: A. M. Brooks Co., 1222 W. Washington Blvd., Los Angeles 7, Calif.

Aurex (Semi-Portable).

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.

Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes

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THE CHEMICAL COMPOSITION OF PERILYMPH IN CATS*†‡§

JULES G. WALTNER, M.D.,
New York, N. Y.

There are few chapters in human physiology as rich in unsolved problems as that of the inner ear. A thorough knowledge of the basic physiology of the inner ear is obviously indispensable if we ever expect to answer the open questions related to otosclerosis, Meniere's disease, and a number of different types of deafness and vertigo.

This study was undertaken in order to establish certain basic facts on the chemical nature of the perilymph in cats and possibly to clarify the question of patency of the cochlear aqueduct.

REVIEW OF LITERATURE

Review of the literature revealed rather meager information on the chemistry and physical properties of the labyrinthine fluids. The relative inaccessibility of the fluid spaces, the very small quantities of these fluids and the considerable technical difficulties in avoiding disturbance of normal physiology during experiments well explain the paucity of information on this subject.

Rossi[1] found the relative viscosity of the perilymph of the pigeon 1.7 as compared with 2.9 of the endolymph. Szasz[2] measured the refractive indices of the perilymph and cere-

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† From the Department of Otolaryngology, College of Physicians and Surgeons, Columbia University, and the Presbyterian Hospital, New York, New York.

‡ This study was assisted by grants from the Research Council of the American Otological Society.

§ Thanks are expressed to Dr. Samuel Raymond, and Mr. Robert Franzl for performing the chemical analysis.

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brospinal fluid of the dog and concluded that the fluids were different.

Ledoux[3] found the refractive index of the perilymph in the cat and in the dog slightly higher than that of the cerebrospinal fluid. In addition, Ledoux noted that after perilymph was removed through the round window, the fluid was soon replaced and successive measurements revealed a progressive lowering of the index of refraction until the index almost reached that of the cerebrospinal fluid.

Aldred, Hallpike and Ledoux[4] and Ledoux[5] studied the osmotic pressure of the labyrinthine fluid of cats and found it higher than that of the cerebrospinal fluid; however, the osmotic pressures of the perilymph and endolymph were nearly the same.

The pH of the labyrinthine fluids in the cat was found to be higher than that of the cerebrospinal fluid (Ledoux[6]).

The chloride, urea N, and the reducing substance contents of these fluids were measured by Ledoux.[5] No significant differences were obtained.

Ledoux[5] mentioned that he found much more proteins in the perilymph than in the cerebrospinal fluid or endolymph of the cat.

One reference was found on living human material. Waltner and Raymond[7] analyzed perilymph from five patients who suffered from Meniere's disease. The protein concentration in perilymph was more than twice that found in the cerebrospinal fluid of the same individual patient. Analysis of endolymph of one patient showed similar quantitative relationship between endolymph and cerebrospinal fluid. The sodium contents of perilymph and cerebrospinal fluid were equal, according to these authors. They concluded that the identical concentration of cristalloids (sodium) and the great difference in protein concentration point was due to a semipermeable barrier between perilymph and cerebrospinal fluid.

As all human material originated from diseased labyrinths (Meniere's disease) it seemed important to obtain more data from healthy animals, the anatomy of which was comparable to the human.

METHOD.

The middle ear of the cat was first exposed and blood, if any, was removed from the tympanic cavity, especially from the area of the round window. A very fine glass capillary was then passed through the round window membrane, and a sample of perilymph was removed with gentle suction. The bony overhang of the niche to the round window was not removed, in order to avoid any traumatism and hemorrhage in the immediate vicinity of the round window membrane. Perilymph was aspirated from both ears in each animal.

Samples of cerebrospinal fluid were obtained by cisternal puncture. The atlanto-occipital ligament was first exposed, and hemorrhage controlled in order to eliminate possibility of blood contamination.

A sample of the animal's blood was taken in each case for blood correction, in case microscopic amounts of blood were found in the perilymph, or in the labyrinthine cavities.

In every second animal cerebrospinal fluid was drawn first, immediately followed by removal of perilymph through the already exposed round window membrane. It was thought that this would reduce the possibility of mixing perilymph with cerebrospinal fluid via the cochlear aqueduct, by greatly diminishing the pressure of the cerebrospinal fluid in the subarachnoidal spaces.

The cat was decapitated on completion of the experiment, and the head was immersed in fixative fluid. The temporal bones and brain *in situ* were studied in serial sections, in order to ascertain possible damage to soft tissues. Tear of Reissner's membrane, or of the basilar membrane, was of special interest, because of the concomitant mixing of perilymph with endolymph. Serial sections were thought to be of importance in obtaining additional check on presence of hemorrhages of microscopic size in the labyrinth.

DESCRIPTION OF HISTOLOGICAL FINDINGS.

Thirty cats were used in these experiments, which were performed under general anesthesia.

Sixteen temporal bones were studied in serial sections. Those animals were selected for serial sections in which the fluids could be removed with minimal or no trauma to tissues, and in which the fluids obtained were macroscopically clear. Description of eight pairs of representative temporal bones is given below.

Group 1.—Histological findings of the ears, in which perilymph was removed first, followed by cisternal puncture.

Cat 814: Samples of perilymph and spinal fluid were macroscopically clear.

There was a clear-cut hole in the round window membrane on both sides. No tear of the membranous partitions of the labyrinth was found. There were a few red blood cells on inner aspect of the round window membrane of the left ear, while the right labyrinth was entirely devoid of free blood. Fibrin-like precipitate filled out most of the cochlear duct of the basal turn on both sides, while a small amount of similar precipitate was visible in some sections of the utricles and saccules. In several sections Reissner's membrane and the basilar membrane were found to be displaced in the direction of the scala tympani. The large vessel, which could be traced as leading to the stria vascularis, was bilaterally engorged especially in the basal turn.

Cat 815: Clear perilymph was obtained on the right side, while the left one was grossly blood-tinged (glass tip broke) and, therefore, discarded. The cerebrospinal fluid was clear.

Right ear: There was a small clear-cut hole in the round window membrane. No tear was found anywhere, but Reissner's membrane was found to be displaced downward in the direction of the basilar membrane in all turns of the cochlea. Few blood cells were present in the vicinity of the round window membrane. Considerable amount of blood was seen in the subarachnoidal spaces and in the cochlear aqueduct, none of which entered the scala tympani. Fibrin-like precipitate was present in the endolymphatic spaces only: cochlear duct, and saccule, utricle and ampullae.

Left ear Extensive tear of the round window membrane and basilar membrane was found, in addition to a heavy hemorrhage in the scala tympani.

Cat 819· Both perilymphs and cerebrospinal fluids were clear A small perforation was found in each round window membrane The basilar membrane was found to be torn bilaterally as a result of sectioning Reissner's membrane was depressed in all turns of the cochlea. Small amount of blood was seen in the scala tympani on both sides, more in the right one

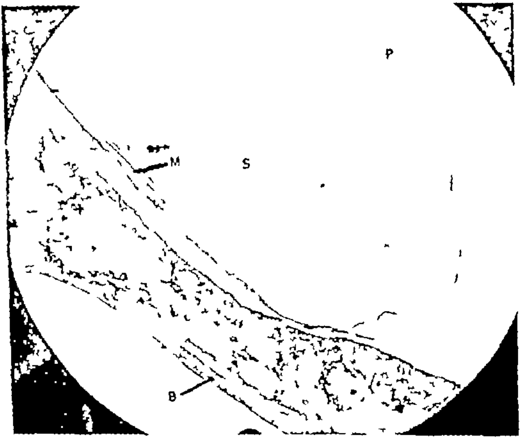

Fig 1 Horizontal section through vestibule S—saccule partially filled with precipitate P—perilymphatic space free of precipitate M—macula of the saccule B—bony labyrinth

Cat 820 The perilymphatic fluids and spinal fluid were clear. A small opening was found in each round window membrane These holes represented one-tenth of the entire window membrane. Fibrin-like precipitate was found in the endolymphatic spaces, mostly in the cochlear duct, some in the saccule (see Fig. 1) and utricle Reissner's membrane was displaced downward in every turn of the cochlea No blood

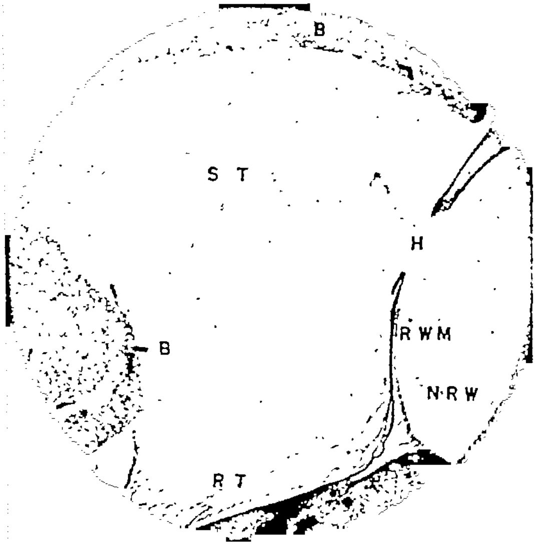

Fig. 2. Horizontal section through the round window membrane. H—perforation of round window membrane made by pipette; RWM—round window membrane; SC—scala tympani free of blood; NRW—niche to the round window free of blood; RT—reticular tissue of cochlear aqueduct; B—bony labyrinth.

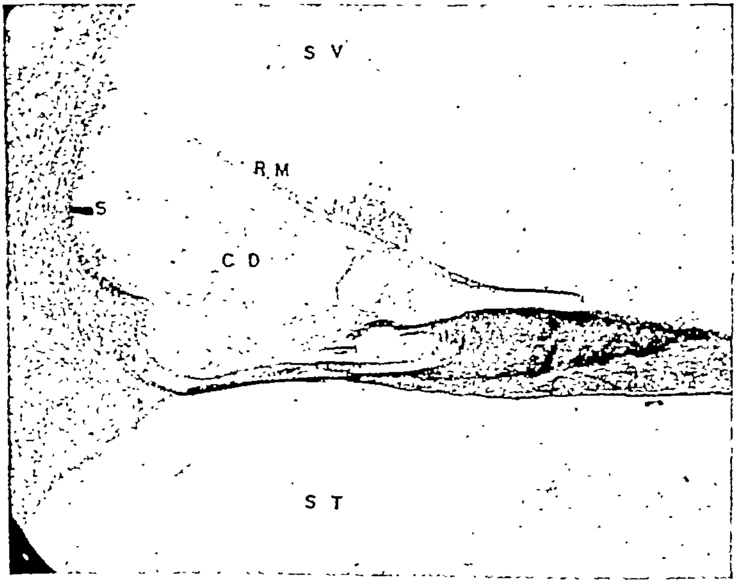

Fig. 3. Horizontal section through cochlea. CD—cochlear duct partially filled with precipitate; ST—scala tympani free of precipitate; SV—scala vestibuli, free of precipitate; RM—Reissner's membrane; S—stria vascularis.

was seen in the right labyrinth, while a few cells were present in the left one.

Cat 821: Perilymph specimens and spinal fluid were clear. Small, clear-cut openings were present in the round window membranes. There were a few blood cells near the opening into the left round window membrane. All membranous walls were intact, but Reissner's membrane and the basilar membrane were displaced in the direction of the scala tympani. The right labyrinth was entirely free of blood (see Fig. 2). Fibrin-like exudate was found in the cochlear duct of the first turn (see Fig. 3), in the saccule and utricle. Few blood cells were seen in the infravestibular portion of the left scala tympani.

Group 2.—Cerebrospinal fluid was removed first, followed by aspiration of perilymph.

Cat 822: Clear samples of perilymph and cerebrospinal fluid were obtained. A small hole was found in each round window membrane. The basilar membrane was torn in most turns, and the labyrinthine capsule was broken. Fibrin-like exudate was present in the cochlear duct. No trace of blood was found in the right labyrinth, while there were about 100 blood cells in the left scala tympani.

Cat 823: Clear fluid specimens were obtained. No tear was found in the membranous partitions. Blood, however, was seen in the scala tympani èxtending into the uppermost turn in the right cochlea. Very little blood was found in the scala tympani of the basal turn of the left cochlea. Fibrin-like precipitate was present in the cochlear duct.

Cat 824: Clear samples of perilymph, but somewhat cloudy cerebrospinal fluid were obtained. A small opening was found in each round window membrane. There were very few blood cells on the left side. No tear was seen on the right side, while the basilar membrane was found to be torn in several of the sections on the left. The labyrinthine capsule was broken on the left; the tear of the basilar membrane, therefore, was considered artefact. Fibrin-like precipitate was present in the cochlear duct and the saccule on both sides. A very small number of erythrocytes were scattered in this fibrinous fluid. The vessel leading to the stria vascularis was greatly engorged (see Fig. 4).

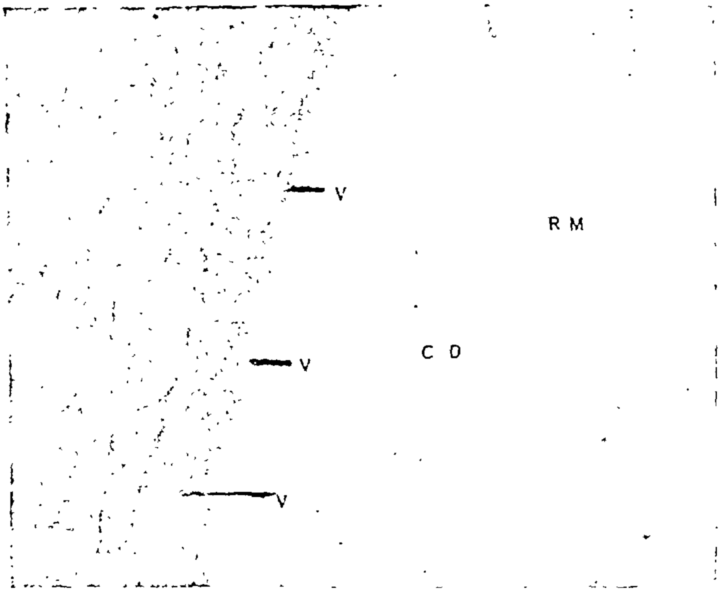

Fig. 4. High power view of cochlear duct. RM—Reissner's membrane; V—strial vessel engorged with blood; CD—cochlear duct containing precipitate with a few blood cells.

SUMMARY OF HISTOLOGICAL FINDINGS.

Fibrinous precipitate was found in the endolymphatic space of each labyrinth except one. In nine labyrinths precipitate was found only in the cochlear duct.

Reissner's membrane was found to be displaced alone, or associated with similar displacement of the basilar membrane in the direction of the scala tympani in every labyrinth. This displacement could not be demonstrated in case the bony labyrinthine capsule was broken during histological preparation.

Engorgement of the large vessel leading to the stria vascularis was clearly visible in six labyrinths.

No blood was discovered in five labyrinths. Few blood cells numbering less than 200 were found near or inside of the round window membrane in ten labyrinths. Rather profuse hemorrhage was seen in the scala tympani of one labyrinth.

CHEMICAL ANALYSIS.

The total amount of perilymph from each ear ranged from 5 to 25 mg. One to 1.5 ml cc. of cerebrospinal fluid was obtained from each animal. In addition, a sample of blood was taken in each case for correction of blood contamination of the perilymph.

Ultraviolet absorption studies of the perilymph showed a peak at 410, a shoulder at 275, and a sharp end absorption beginning at 265 millimicrons. The peak at 410 should be considered due to hemoglobin contamination. The hemoglobin absorption of the blood sample taken in each individual animal was compared with the hemoglobin absorption of the perilymph, and thus a quantitative measure of the amount of blood contamination was obtained. The shoulder at 275 was considered to be due to tryptophane-tyrosine content of the protein present. Purines and ascorbic acid, however, absorb at this wave length, and were probably present in the fluid (see Table I).

TABLE I—ULTRAVIOLET ABSORPTION OF PERILYMPH IN CATS

Cat	Conc. %	D(275)	D(410)	Blood $\frac{D275}{D410}$	Corrected E (1% 1 cm.)
*814	Rt. 16 3	142	013*	49	008
	Lt 9 9	132	030		012
816	Rt 3 9	.327	212	.38	028
	Lt. —	—	—	—	—
819	Rt. 13 1	1 04	1 68	40	028
	Lt. 5 0	182	165		024
*820	Rt. 8.8	133	017*	41	014
	Lt. 19 6	448	458		013
*821	Rt. 20 5	.200	020*	37	010
	Lt. 11 7	197	078*		014
822	Rt. 7 6	450	578	44	026
	Lt. 5 6	580	800		041
823	Rt. 12.2	630	1 03	40	018
	Lt 11 5	518	778		018
*824	Rt. 4 8	142	026*		027
	1 Lt 8 1	510	680	45	026
	2 Lt. 5.8	345	500		020

In specimens marked * there was no microscopic evidence of blood in the labyrinth.

The spectrum of the cerebrospinal fluid revealed only a slight shoulder in the rise of the curve to the end absorption at 265 (see Table II). It is evident from the data that there are marked quantitative differences in the absorbing substances present in the perilymph and cerebrospinal fluid.

TABLE II—ULTRAVIOLET ABSORPTION OF SPINAL FLUID.

Cat	Conc. %	D(275)	D(410)	Blood $\frac{D275}{D410}$	Corrected E (1% 1 cm.)
814	100	.458	.097	.49	.004
815	8.8	.086	.021	.38	.009
819	100	.318	.023	.40	.003
820	12	.063	.012	.41	.005
821	100	.376	.022	.37	.004
822	10	.147	.047	.44	.012
823	100	.533	.122	.40	.005
824	6.6	.090	.010	.45	.013

In Tables I and II the ultraviolet absorption is given, together with the extinction coefficients E (1 per cent 1 cm.) Correction was made for the blood contamination.

TABLE III—TOTAL NITROGEN IN PERILYMPH OF CATS.

Cat	Weight (mg.)	Total Nitrogen Microgram	Blood Correction (Mg. %)	Corrected N (Mg. %)
814	Rt. 17	11	0	67
	Lt. 16	13	1	73
815	Rt. 7.7	3.9	6	45
819	Rt. 13	14.0	21	85
	Lt. 5.0	2.4	5	42
820	Rt. 8.8	2.9	0	33
	Lt. 21	5.9	4	24
821	Rt. 23	6.5	0	28
	Lt. 12	5.0	0	41
823	Rt. 14	6.4	15	30
	Lt. 13	5.2	12	29
824	Rt. 7.2	5.4	1	73
	Lt. 8.2	7.1	19	68
822	Rt. 7.6	9.6	16	110
	Lt. 5.8	5.1	29	68

After completion of ultraviolet measurements perilymph and cerebrospinal fluid were digested with Kjeldahl mixture. The ammonia in the digest was measured by the microdiffusion method of Conway. Correction was made for blood N in the fluids (see Tables III and IV).

TABLE IV—TOTAL NITROGEN IN CEREBROSPINAL FLUID.

Cat	Weight (mg.)	Total Nitrogen Microgram	Blood Correction (Mg. %)	Corrected N (Mg. %)
814	140	21	0	15
815	88	14	1	15
819	98	11	0	11
820	117	4.8	4	4
821	125	21	0	17
822	17	5.8	1	33
823	121	19	0	15
824	66	5.5	0	8

DISCUSSION.

The presence of fibrin-like precipitate in the endolymphatic spaces in all specimens except one is interpreted as sudden increase in production of endolymph. Marked engorgement of the vessels which lead to the stria vascularis and occasional blood cells in this precipitate (Cat 824 Rt.) point to close relationship between secretory activity and vascular supply (see Fig. 4). There was no evidence of similar precipitate in the perilymphatic spaces (see Figs. 2, 3) which, especially the scala tympani, were primarily involved in the experimental perforation of the round window membrane. Presence of the same precipitate in the saccule (see Fig. 1), utricle and ampullae of the semicircular canals is even more certainly explained by secretory activity, because they are located quite far from the round window membrane, the place of the only injury sustained by the labyrinth *in vivo*. Finally no precipitate was formed in the semicircular canals, the only part of the endolymphatic spaces which does not contain any histological structure capable of secretory activity.

Although no direct evidence has ever been obtained on the location of the source of endolymph, the stria vascularis has been generally accepted as the site of such function (Shambaugh, etc.[2]). The above described histological findings in these experiments are strongly suggestive that similar secretory activity should be ascribed to the maculae of the utricle and saccule, and the cristae of the semicircular canals. Saxen[9] came to a similar conclusion on studying the histology of congenital deafness. Histological findings of Nachlas and Lurie[10] on the stria vascularis of Dalmatian dogs also pointed to the region of the maculae, cristae and the bulb of the endolymphatic duct as possible source of endolymph.

The greatest secretory activity was in evidence in the cochlear duct of the first turn of the cochlea, which is nearest to the round window membrane. The increased secretory flow is interpreted as an effort to equalize the sudden loss of perilymph, which was caused by aspiration through the round window membrane. Replacement of perilymph through the membranous walls of the cochlear duct is possible. Permeability of Reissner's membrane for cristalloids was proven by Gisselsson[11] and Altmann and Waltner.[12] Histological evidence of these experiments, therefore, suggests that perilymph may be replaced by fluid produced in the endolymphatic spaces.

No data were found in these studies which gave any support to the theory of free intercommunication of fluids between cerebrospinal fluid and perilymph. In a few instances blood penetrated from the subarachnoidal spaces into the cochlear aqueduct without reaching the scala tympani of the cochlea. Were the cerebrospinal fluid the major source of perilymph, blood would have been carried into the cochlea by the flow of cerebrospinal fluid.

The minute amounts of perilymph obtainable, and the unavoidable contamination of some of the samples with microscopic amounts of blood, make caution necessary in drawing conclusions based only upon the chemical analysis of the perilymph. Microscopic studies of the ear, in serial sections, in addition to two different analyses of the fluids, ultraviolet absorption and total N, improve the reliability of the conclusions drawn.

It would seem that the amount of blood contamination could be greatly reduced by aspirating perilymph in the dead animal after blood circulation has stopped, but such procedure would introduce numerous unknown factors; therefore, no reliable conclusions could be drawn as to the normal physiology of the perilymph.

Studies in serial sections failed to reveal any blood in five labyrinths (814 Rt., 820 Rt., 821 Rt., and Lt., and 824 Rt.). These findings fit perfectly into the data of the chemical analysis, and confirm the validity of measurements in other samples slightly contaminated with microscopic amounts of blood. Parallelism between miscroscopic findings and chemical analysis existed to a great degree. A good example is Cat 820; a few blood cells were found under the microscope in the left labyrinth, while there was no evidence of blood in the right labyrinth, thus confirming the ultraviolet measurement. The greatest difficulty in the chemical analysis, contamination of very small samples of fluid by blood, was thus reduced to minimal importance.

Studies in serial sections proved to be very helpful and indispensable to rule out accidental mixing of perilymph with endolymph through an artificial tear of the membranous labyrinth.

The great difference between the protein contents of the perilymph and cerebrospinal fluid disproves previous concepts of identity of these two fluids. In the light of the same data, a free flow of cerebrospinal fluid through the cochlear aqueduct into the scala tympani is equally untenable.

No significant difference in the protein contents of perilymph was found between samples taken before or after removing cerebrospinal fluid.

In a few instances the sodium content was measured with a flame photometer, and was found to be almost identical in concentration in the perilymph and cerebrospinal fluid. Sodium was selected for measurements because it stays mostly in the interstitial tissue fluids; thus it is most likely to take part in diffusion or ultrafiltration.

The greater concentration of protein found in the perilymph of humans suffering from Meniere's disease, as compared with the cerebrospinal fluid of the same patients, is most probably not pathological in nature. Similar quantitative differences exist between the cerebrospinal fluid and perilymph of normal cats.

Further study of the nature of human labyrinthine fluids would offer valuable information. Ectasia of the endolymphatic spaces in Meniere's disease certainly points to a disorder of the fluid system. A sizable group of unexplained cases of deafness, especially those with sudden onset, may well benefit from better knowledge of the labyrinthine fluids.

SUMMARY.

1. Measurements of the ultraviolet absorption and total nitrogen were made on the perilymph and cerebrospinal fluid of 30 cats.

2. The total N concentration was more than twice that of the cerebrospinal fluid taken from the same animal in every instance.

3. Microscopic studies of sixteen labyrinths were done in serial sections. There was no evidence of microscopic blood contamination in five labyrinths, while only a few blood cells were present in ten.

4. Blood correction in the chemical analysis proved to be accurate when data were compared with microscopic findings.

5. Mixing of the perilymph with endolymph was ruled out by microscopic studies.

6. The great difference in chemical composition between perilymph and cerebrospinal fluid rules out all possibility of free flow of fluid between subarachnoidal and perilymphatic spaces.

7. The greater concentration of proteins in the perilymph of humans suffering from Meniere's disease is most probably not pathological in nature, in view of the above findings.

8. Histological studies of the same animals point to the increased secretory activity in the endolymphatic spaces following drainage of the perilymph through the round window

membrane. This again is interpreted as indirect evidence of the role of the endolymph as source of perilymph.

9. Localized precipitation of endolymph in the cochlear duct, in the saccule, utricle and ampullae of the semicircular canals is considered as indirect evidence of increased production of endolymph in these anatomical structures.

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THE INARTICULATE CHILD: A PROBLEM IN DIAGNOSIS.*

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Although the title of this paper might suggest that the approach to the inarticulate child is academic and no more, that inference would be entirely incorrect. The state of these children is always distressing, and so is the state of their parents. The approach to the problem, for a problem it is, while it must be compassionate, must also be scientific. Whether these children, in spite of their handicaps, are to lead happy, successful and useful lives depends, first of all, upon a precise determination of the nature of their handicaps. An inarticulate child is not necessarily deaf, nor is he necessarily dumb, in the dictionary sense of being unable to speak or in the more colloquial sense of the term. He may be aphasic because of brain damage or for other reasons; he may not talk, or his speech may be incomprehensible for psychological or functional reasons.

How the inarticulate child shall be treated depends upon the identification of the special cause of his inarticulateness, What his future life is to be depends upon how he is treated. An enormous responsibility, therefore, rests upon the otologist when once the parents bring the child to him. I might go further and say that an equally enormous responsibility rests upon the general practitioners and the pediatricians, who so often see these children first, and by whose advice the parents are guided in what they do next, and how soon they do it.

The inarticulate child is more frequently encountered today than he ever was before, for two reasons: first, that more newborn children are surviving the difficulties of birth and of early prenatal life. Rh incompatibility in the parents is approaching control. Maternal toxemia and the hemorrhagic

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complications of pregnancy are also approaching control. The child himself, thanks to improvements in therapy, including modern antibiotic therapy, is surviving such illnesses as encephalitis and meningitis, which formerly had a very high mortality and which still leave residua, among which deafness is prominent.

The second reason why we are seeing more inarticulate children today is that they are being referred to us with much greater frequency. Some errors are still being made, but I am glad to say that the day has almost passed when pediatricians, general practitioners, and even otolaryngologists would tell the parents that a child is too normal otherwise to have anything wrong with his hearing, or that he will probably outgrow his unawareness of sound, or that, if his hearing is really impaired, he must be five or six years old before auditory testing is possible. It is never too early to start testing and training the deaf or otherwise inarticulate child. It is also never too late, but the end results are less good than when the start is made early. For one thing, the child whose impairment is slight or even moderate can frequently be so rehabilitated as to keep up with normal children. Schools for the deaf serve an invaluable purpose, but a normal life with normal children is better. For another thing, as Ewing and Ewing[1] emphasize, interest in persons is an essential foundation of the acquisition of lip reading and speech training. It is, therefore, far better that such training should begin while the child is still in close physical contact with his mother and his nurse, and has not yet become deeply concerned with physical things.

In recent years there has been a great deal of active emphasis on testing of the hearing of infants and young children. This is as it should be. Our methods, however, still leave something to be desired. One might gather, from the medical literature and from presentations at medical meetings, that the only techniques which provide satisfactory diagnostic information are such complicated ones as psychogalvanic skin-resistance audiometry (PGSH), electroencephalography, or pure tone audiometry by peep-show methods. This is far from the truth.

These are excellent tests, but only in their places; they are not infallible, they are merely adjuncts to more basic procedures. The equipment is expensive, and special training is required to operate it. These are valuable methods in speech and hearing centers, but they are not suitable for use in the average otolaryngologic practice, and they are, of course, completely impractical for use in the office of the general practitioner who, either because of desire or necessity, combines otolaryngology with general practice. Finally, they are not methods in any way suitable for use in testing very small children.

One of the most unfortunate results of the impression that these diagnostic methods are necessary for diagnosis, is that so many physicians apparently believe it necessary to refer the inarticulate children to speech and hearing centers in which they are available. Lest I be misunderstood, I hasten to say that I have much to do with the operation of the speech and hearing center at the Tulane University School of Medicine, where equipment for these tests is available, but where they are seldom carried out on very young children.

A second unfortunate result of the impression that these tests are essential for diagnosis in children is the adoption of this point of view by residents in otolaryngology. It is the same sort of error, of course, which leads residents in other specialties, who all too often have the bad example of their elders to guide them, to substitute laboratory tests for the use of simpler clinical methods, often to the detriment of their patients. Residents of otolaryngology, like the other residents, must be brought back to first principles. In the diagnosis of hearing and speech difficulties, these principles are, in this order, history, clinical observation, and clinical evaluation, with testing last of all.

DIAGNOSTIC CONSIDERATIONS.

For a long time it was assumed that hearing tests which had been found satisfactory for adults were equally satisfactory for children. They are not. As Myklebust[2] has well expressed it, the evaluation of hearing in adults who are emotionally, intellectually, physically and perceptually mature, is a very different process from its evaluation in children who are none of these things. An experience recorded by Ewing and Ewing[1] makes this clear. They found that three children who were apparently totally deaf when tested with loud sounds, responded to instructions spoken in a moderately loud voice at a distance of six feet.. Obviously, factors other than the purely physical ones of pitch, loudness and duration of sound may determine the young child's response or failure to respond to it. Obviously, the whole basis of tests of hearing in young children requires re-evaluation.

Pure tone audiometry has proven an extremely useful method for diagnosing hearing losses in adults. In children, as most of us realize, pure tones are not suitable auditory stimuli. The child does not readily respond to them, because they are beyond his perception. He is much more likely to respond promptly to mixed sounds at the same level of intensity, or even at lower levels. My own experience agrees with that of other observers: reliable responses to pure tones are unlikely to be achieved until the general mental and perceptual level is that of the average normal five to six-year-old child. The important consideration here is that five or six years of age is far too late to diagnose a hearing loss or speech difficulty and to do something about it.

Modifications of pure tone audiometry are also unsatisfactory in young children. The PGSR test has two objections: first, that it is dependent upon pure tone stimuli, and these, as has just been pointed out, are not reliable at this age; second, the technique has not been standardized on young children with normal hearing, and there are, therefore, no criteria to evaluate hearing losses in abnormal children. Still another objection might be mentioned, that if the child is emotionally disturbed any test that depends upon a conditioned reflex to a disagreeable stimulant may indicate a hearing loss when none is present, or a greater hearing loss than really exists.

When the peep-show technique of pure tone audiometry[3] is used in children over five years of age, the results are satisfactory, and a threshold test can be quickly run. Between the ages of three and five years, however, the technique, ingenious as it is, is not entirely reliable, and the results are not commensurate to the time spent on it. Below the age of three years it has been completely useless in our hands.

As to the electroencephalograph, it is of undeniable value in revealing brain damage or in excluding it. Special techniques have been developed for use in children when they are asleep,[4] but we prefer to use it with noise makers in a free field. It often helps to explain delayed, retarded or unintelligible speech in a child with no hearing loss or only slight loss. It remains, however, an adjunct method, which does not supplant basic clinical principles.

Diagnosis in the preschool child is time-consuming and frustrating. More often than not, as Bordley[5] says, it results in the otologist's being more hopeful that he is right, than being sure that he is. The more carefully the investigation is conducted, however, the more likely he is to be right.

Before he attempts to diagnose hearing or speech difficulties in young children, the otologist must have clearly in his mind the stages of normal development:

The normal child sits alone at five to seven months and crawls between eight and ten months, unless he skips the crawling stage and begins to walk at once. Ordinarily, however, he walks between 12 and 18 months. Toilet training for the bowel occur between 15 and 24 months, and for the bladder between 18 and 24 months during the day, and between 24 and 30 months at night if he is taken up.

If hearing develops normally, it will be according to the following schedule:[6]

There is some response to sound immediately after birth, which becomes more definite at one month. The response takes the form of a reflex, which is usually palpebral. During the first three months of life infants respond more readily to noise makers than to the voice.

During the fourth through the sixth months the voice is at least as good as a noise maker as a form of auditory stimulation. A quiet voice is more effective than a loud voice.

During the second half of the first year of life the child continues to respond to noise by reflex reactions. He becomes adept at recognizing meaningful sounds, such as tapping on the nursing bottle. He begins to localize sound by turning his head or his eyes to the source. Certain voices quickly attract his attention.

During the second year of life the child responds to simple speech tests. The whispered voice is unsuitable for testing at this age, and a quiet voice continues to attract his attention more quickly than a loud voice.

During the third year of life the child shows a great capacity to localize sound. Speech and voice tests are now a satisfactory method of testing hearing.

Between the ages of three and five years the normal child responds to vocalizations and whispers; he will also respond to pure tone audiometric testing, though as a rule at least another year must pass before this technique gives entirely satisfactory results.

The normal child, if a girl, will usually begin to say single words between 10 and 12 months. A boy does not usually begin to talk until several months later, when he is 14 to 16 months. Both use two-word sentences between 18 and 24 months.

It is now known that during the first 12 to 18 months of life there is no difference in the vocalization of the normal child and the deafened child, even when the deafness is severe. The deaf child's voice has natural quality and inflections, and skilled training, if begun early, can do much to conserve the natural properties.

It is extremely important that the otologist should thoroughly understand these normal stages in the child's development. It is equally important that he realize that the times just stated vary widely in individuals. On the other hand, the child who has not said a single word up to the time he is 16 months old, or who has showed no response to sound by the time he is 12 months old, needs medical consultation, and needs it promptly. The first three or four years of life are the critical ones in the development of any child, and they are even more important in the child whose speech and hearing are impaired to any degree at all.

ROUTINE OF INVESTIGATION.

To turn now to the positive side of the problem, the investigation of the inarticulate child begins with a complete prenatal and postnatal history, careful observation, evaluation of the findings, and then, last of all, actual testing of the hearing. All of this takes time, and nothing is gained by trying to make haste or to use short cuts; furthermore, the assistance of the mother is imperative in the investigation, and she will withdraw into herself if the physician is not considerate, thoughtful and sympathetic. Incidentally, when the investigation is concluded, he must take the time to explain the situation to her in the simplest possible manner, and to give to her as much hope as the child's condition warrants.

History Taking: Our own procedure, when an appointment is made for an inarticulate child, is to defer it for several days, in order to give the parents time to fill out the questionnaire, which is immediately mailed to them, and to return it to us for review and analysis before the appointment. It is sometimes difficult to secure an exact statement from the mother as to the child's difficulties when the question is abruptly broached to her at the office, nor is the other information she supplies always prompt and accurate. Given the time, however, she will clarify her own thoughts by writing down the replies to the questions asked in the history sent to her, and the data will be as full as can possibly be secured.

If no appointment has been made, a short history form is used in the office at the first visit, but it is not so satisfactory.

The questionnaire mailed to the mother provides for detailed information concerning the family history; the prenatal history; the delivery; the diseases, operations and accidents of postnatal life; and the story of the child's development and general progress, as well as his progress in speech and hearing. Specifically, the following points are covered, in addition to details of the family history and maternal history: When the speech or hearing difficulty was first noticed. Precisely what form it takes. Whether other difficulties are also present, such as visual impairment or behaviorism. The weight and respiration at birth. Feeding problems. All the possible illnesses of childhood and their complications. When the child first sat, walked, fed himself, was toilet-trained, dressed himself, said words and made sentences. Handedness. Appetite.

Habits of sleep, including bed-wetting. Other habits, such as sucking the thumb and biting the nails. Temper tantrums and destructiveness.

Questions are asked as to how the child compares with other children in progress and behavior, whether he can convey his meaning, whether he can understand his mother, whether he prefers the father or the mother, and how he reacts to people in general. In short, there is almost no conceivable piece of information which this form does not provide for, and parents are usually most cooperative and helpful in filling it out.

Clinical Observation: When the child is brought to the office, all the circumstances are kept as normal as possible. The young child may have recollections of other visits to a physician's office and of unpleasantness associated with them, and such memories would hinder the investigation if they were brought back to him. The physician, therefore, removes his head mirror and replaces his white office coat with his ordinary coat. After a casual greeting, the child is allowed to move about as he pleases while the physician discusses with the mother the data she has supplied, or while a brief office history is taken if the longer form has not been filled out. During this time the child is unobtrusively observed.

The hearing of young children can be tested in various ways. Our own preference is for the use of noise makers in a free field. The equipment which we use, and which is purchased from a music supply house, makes sounds between the 200 and 5,000 frequencies. The noise makers are checked as to frequency or frequency range with an octave analyzer on a sound level meter, after which it is possible to produce sounds of the decibel or power level desired and to record the results accurately. We begin with a tone around the 25 to 30 decibel level, since it has been established that many children will respond to a tone at this level and not to one at a higher level. If no response is elicited, the sound is increased until it is heard or it is clear that no sounds can be heard.

As has been pointed out, we try to make the conditions of the test as natural as possible. For all practical purposes, testing is by the so-called cessation of activity technique, which, as the name implies, means that the child ceases whatever he has been doing because a sudden noise is impressed upon his consciousness. The windows in our office overlook the Mississippi River, and there is a great deal of industrial and shipping activity on it at all times. Testing can often be carried out while the child is absorbed with the many sights along the river. If he is not interested in these sights, or is too small to be interested in them, he is given some type of toy, most often a ball, to occupy him. The very young child is permitted to have his bottle.

Eventually a detailed investigation of the nose, nasopharynx and Eustachian orifices must be carried out with the electropharyngoscope, but this is always deferred until all other clinical observations have been made. If it were the first phase of the investigation it would obviously end all hope of rapport with the child. Laboratory tests of hearing are also deferred until clinical observations are concluded.

DIFFERENTIAL DIAGNOSES.

The differential diagnosis of inarticulateness is usually possible by these simple clinical methods if enough time—it takes a great deal—is devoted to them, and if the observation is careful and thorough. All of the facts which are stated hereafter, can be secured in this manner.[6] When they are properly evaluated, the cause of the child's failure to speak, or of his poor speech, is usually evident.

Response to Sound: The child with impaired hearing responds to sound consistently at the threshold level and age level. If he can hear a sound at 30 db or less, or if he can hear conversational speech, his inarticulateness cannot be attributed to inability to hear.

The child who does not respond to sound until it falls between 30 to 60 db is suffering from a moderate hearing impairment, frequently of conductive origin, and usually correctible. Until the correction is made, however, he cannot acquire normal speech because he does not hear normal conversational tones. If the loss is no more than 40 db, and he is taught lip reading, he can remain in regular schools provided with special seating arrangements and similarly aided.

If the child responds to sounds from 60 to 100 db, his hearing loss is probably on a perceptive basis, and speech can be heard only if it is amplified. If there is no response to sounds of less than 100 db intensity, the hearing loss is extremely serious. Hearing aids will be of no value and schooling in special schools for the deaf will be required.

In contrast to the child with impaired hearing, the aphasic child does not respond to sound consistently. The emotionally disturbed child is apparently willful in his failure to respond. The mentally deficient child responds consistently to the extent permitted by his mental capacity; his response is usually to sounds which indicate food and similar things to him.

Visual Clues: The child with impaired hearing relies heavily on vision and is, therefore, unduly sensitive to hearing and other visual clues. The aphasic child, because of central nervous system damage, either does not rely on vision at all or does so erratically and intermittently. The emotionally disturbed child does not rely on vision, since his disturbance is generalized in effect. The mentally deficient child need not rely on vision, for he is using his auditory sense to his full capacity, and, therefore, has no loss for which to compensate.

Speech: The child with impaired hearing acquires a speech pattern consistent with his capacity to hear. He does not vocalize for pleasure but uses sound with intent and purpose, in a logical manner, to express his feelings and needs. Echolalia (parrot or echo speech) is not present. The impairment of the quality of the voice depends upon the hearing loss.

The aphasic child attempts to speak, but the result is jargon, which is spontaneous but meaningless. He uses his voice playfully or indiscriminately, without meaningful intent or purpose. Echolalia may be mild, moderate or marked. The tonal quality of the voice remains about normal.

The emotionally disturbed child seldom speaks, and is frequently completely mute. He does not usually use his voice for pleasure or to attract attention, though in less severe cases the voice is sometimes used in play, but always erratically. He does not use jargon. Echolalia is present in severe emotional deviations. The tonal quality is normal.

The mentally deficient child acquires speech eventually but at a later age than normal. He uses his voice for pleasure and projectively at the level of his mental capacity. Echolalia is not present. The tonal quality does not deviate from normal.

Use of Gestures: By the age of two years the child with impaired hearing uses gestures himself and understands those of others. The aphasic child does not use gestures effectively. Sometimes he is unable to make them. In other instances he uses them frequently but inconsistently. The emotionally disturbed child, who has rejected his environment, does not use gestures at all. The mentally deficient child uses them in accordance with the stage of his mental development.

Laughing, Smiling, Crying: The child with impaired hearing smiles and laughs less than the normal child. He cries often, and with vigor and feeling. The aphasic child does not laugh or smile freely, though he may laugh compulsively, in a defensive manner. His cry is typically a whine. The emotionally disturbed child does not laugh or smile except in a preoccupied or disturbed manner. He does not share the laughter of others. He cries without obvious reason. The mentally deficient child laughs, smiles and cries normally so far as tone is concerned, but his reactions are delayed.

Other Senses: The child with impaired hearing has no disturbance of visual perception, and the same is true of the emotionally disturbed child. The child with aphasia frequently misperceives both objects and sounds. The mentally deficient child frequently misperceives sounds but not objects, his perception being, on the whole, commensurate with his mental capacity.

The child with impaired hearing is usually sensitive to tactile sensation, relying on his sense when the visual sense is in use. The aphasic child is not unduly responsive to tactile stimuli and tends to ignore them while he is engaged in activity. The emotionally disturbed child is generally unresponsive to such stimuli. He acts as though tactile sensations were nonexistent. The mentally deficient child is not unduly responsive to them, since he is using all his senses to the best of his ability.

Motor Behavior: The child with impaired hearing sits and walks normally at the usual age. He is hyperactive, but his activity is consistent and meaningful. His balance may be poor and he tends to shuffle as he walks. The emotionally disturbed child may be late in sitting and walking because he does not want to sit or walk. He may show stereotyped activities, such as rocking or twirling. The mentally deficient child is slower in sitting and walking than children in the other groups and is retarded in all other motor activities.

Attention to Facial Expression of Others: The child with impaired hearing is unduly responsive to the facial expressions of his associates. The aphasic child is incapable of the sustained attention necessary to profit from them. The emotionally disturbed child, as a manifestation of his rejection of the world, ignores face to face conduct. The mentally deficient child is realistic to the extent of his capacity to profit from attention to the facial expressions of others.

Emotional Expression and Development: The child with impaired hearing is immature and delayed emotionally, but is otherwise normal in this respect. The aphasic child is immature emotionally. Such emotions as he displays are superficial and he has no basic means of expressing them. The emotionally disturbed child does not show normal response to affection. He does not laugh, cries very little, and apparently has no ability to express emotion. The mentally deficient child is more immature emotionally than the child with impaired hearing. His emotional expression is in proportion to his mental age.

Social Perception: The child with impaired hearing is somewhat less efficient in social perception than the normal child. He relies entirely on vision. He is normally shy and reticent. The aphasic child, who is incapable of understanding social situations, attempts to relate them to his environment. He has no shyness or reserve. The emotionally disturbed child behaves as he pleases. He regards people as objects and does not include them in his world. The mentally deficient child is aware of people around him and accepts them at the level of his mental capacity.

Perseveration, Distractibility, Disinhibition: These qualities are not observed in the child with impaired hearing. They exceed normal expectation in the emotionally disturbed and the mentally deficient child, and are very marked in the aphasic child.

SUMMARY.

The inarticulate child is being referred to the otologist more frequently than ever before, but the belief persists in many quarters that he must be investigated by elaborate laboratory methods. Actually, pure tone audiometry, PGSR audiometry, audiometry by the peep-show technique, and electroencephalography are not suitable for diagnosis of speech and hearing defects in young children. Instead, diagnosis is accomplished, and in this order, by detailed history-taking, clinical observation, and clinical evaluation, with laboratory investigation last of all. The use of noise makers in a free field, by the so-called cessation of activity technique, is a desirable method of testing hearing in young children because it can be done unobtrusively, in natural circumstances.

A technique of history-taking, auditory testing and clinical evaluation of the total child is presented, and the criteria of differential diagnosis are briefly outlined.

The approach to the problem, while it is entirely scientific, does not forget that both the child and his parents are human beings in distress, who need all of the physician's understanding and compassion in addition to his technical skill.

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SYMPOSIUM:

BRONCHOGENIC CARCINOMA.

I—SUSPICION.*†

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In preliminary summary, certain general facts pertinent to the subject of bronchogenic carcinoma are deserving of comment. There is evidence that the actual incidence of the disease appears to be increasing, particularly in men. Recent advances in thoracic surgery, yielding low mortality rates, render exploration of the chest and surgical treatment of the disease by excision both safe and effective. The ability to cure patients suffering from bronchogenic carcinoma, however, depends upon prompt surgical treatment; thus, early recognition based upon suspicion yields the most productive results in the control of bronchogenic carcinoma. Herein lies the great opportunity for the physician who first sees the patient, whether he be otolaryngologist, internist, or general practitioner.

Unfortunately, there are a number of factors which interfere with successful treatment of bronchogenic carcinoma. These could be classified as the factor of the disease itself, the factor of the patient, and the factor of the physician. The factor of the disease itself can be illustrated in the following fashion. While it is a recognized fact that early bronchogenic carcinomas are generally discovered in routine thoracic roentgenograms and that their successful treatment has followed such discovery, even under serial roentgenographic observation hopeless situations have arisen. The following report of a case is cited:

Case 1: A 63-year-old furniture factory worker and former miner had been examined over a period of two years by the chest clinic of a city

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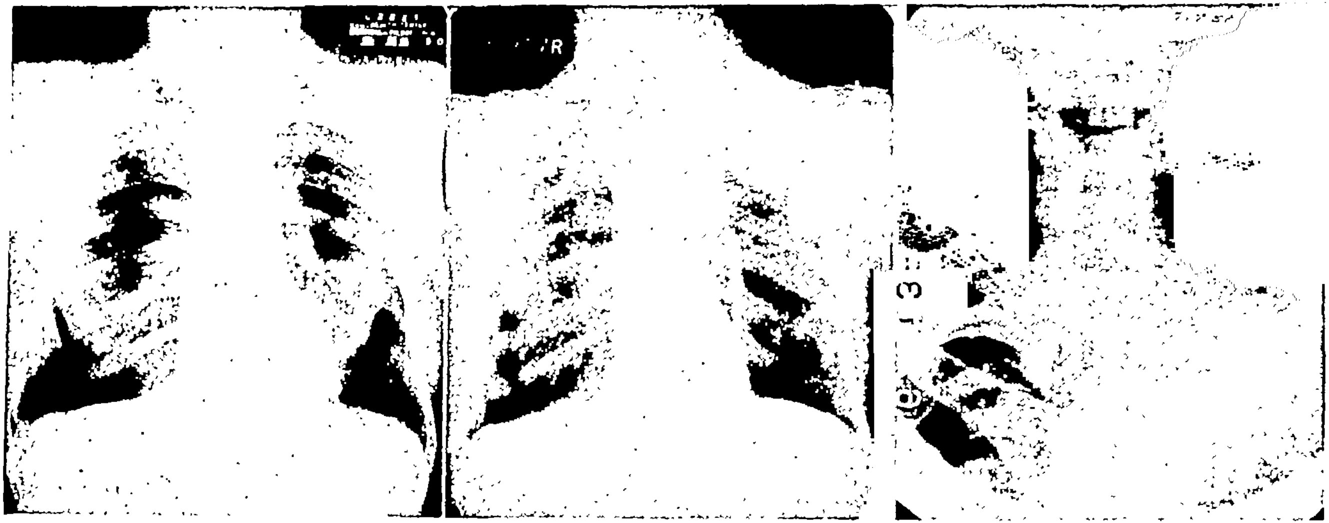
(a) (b) (c)

Fig. 1: a—Chest film of a 63-year-old man showing bilateral apical lesions considered to be consistent with the diagnosis of silicosis. This film represents the last of four films taken over a period of two years, all of which demonstrated stability of the apical lesions. b—Subsequent film of same patient revealing a large left apical density. This film was made less than one year after that shown in a. c—Anteroposterior view of cervicothoracic spine of same individual. A destructive lesion of the left second thoracic transverse process is shown.

Public Health Service. Typical nodular lesions of silicosis were found in the X-ray film of the chest (see Fig 1a), and repeated X-rays eventually revealed stability of these lesions. The last film was taken three months prior to the onset of the patient's illness, which was ushered in by pain in the left forearm and hand, and later by the development of a left Horner's syndrome. A roentgenogram taken several months following the onset of the illness revealed a left apical intrathoracic tumor, (see Fig. 1b); and in a film of the spine destructive lesions of the transverse processes and bodies of the first and second thoracic vertebrae were seen (see Fig. 1c). In an attempt to arrive at a diagnosis, a supraclavicular and an axillary lymph node were removed. Neither of these nodes manifested abnormalities which would suggest the nature of the left apical tumor. Successful biopsy, however, was performed by means of a needle inserted into the region of the transverse process of the first thoracic vertebra. Microscopic examination of the tissue removed revealed a well-differentiated squamous cell carcinoma. The findings in this case were consistent with the diagnosis of a superior sulcus tumor that was inoperable because of neural and osseous involvement.

It is highly unlikely that any available methods of the present day could have led to a satisfactory outcome in the case of this particular patient. One cannot say that unavoidable delay existed in the diagnosis.

The factor of the patient in terms of an interfering factor in the successful treatment of bronchogenic carcinoma can be expressed as follows: Either the patient fails to seek medical aid in the presence of evident symptoms, or he refuses to follow worthy advice. Convincing evidence comes from a series of ten patients reported on by Abeles and Ehrlich.[1] All of these patients had solitary undiagnosed pulmonary lesions discovered in routine thoracic roentgenograms. All had no symptoms and all refused exploratory thoracotomy. Within a year of observation five of the group exhibited evidence of spreading intrathoracic malignancy.

The third interfering factor is that of the physician himself. Countless examples can be found to illustrate that physicians unintentionally have followed a program of watch and wait rather than leading patients directly to definite diagnostic steps, even to the extent of exploratory thoracotomy. In the course of the past ten years two factors have become widely recognized; namely, that conventionally recorded signs and symptoms of bronchogenic carcinoma are of little value in the recognition of the disease in its early phase, and that pulmonary cancer is the only visceral malignant lesion which casts a shadow in an ordinary roentgenogram. The foregoing two points indicate action consisting of encouraging patients to have X-rays of the chest made at intervals of at least once a year or even more frequently. Obviously there is a certain practical limit. In the management of adult patients it is not unreasonable to insist upon X-rays at annual intervals and X-rays of the chest whenever respiratory symptoms appear.

There are certain situations in which lie opportunities for early diagnosis of bronchogenic carcinoma. Some of these can be listed as follows: The solitary circumscribed pulmonary lesion, the patient with hemoptysis, the patient with pneumonia, and the patient in whom one discovers a localized wheeze. Because of the frequent occurrence of inflammatory pulmonary lesions resulting from bronchial obstruction due to bronchogenic carcinoma, an illustrative report of a case follows.

Case 2: A 62-year-old white carpenter was first admitted to the hospital with the complaint of cough, hemoptysis and dyspnea for ten days. The patient was febrile and a roentgenogram (see Fig. 2a), disclosed changes consistent with atelectasis in the lower portion of the right upper lobe. On treatment consisting of penicillin intramuscularly the patient's symptoms rapidly disappeared. A roentgenogram taken approximately two weeks later revealed persistence of the lesion in the lower portion of the right upper lobe. Because of failure of this lesion to clear and because of persistence of cough, bronchoscopy was advised.

This procedure revealed no evident bronchial lesion. Cytologic studies of the sputum revealed no evidence of neoplastic cells, and no organisms of tuberculosis were seen in specimens of sputum. Skin tests for coccidioidin and histoplasmin were negative and a tuberculin reaction was positive in the second strength. Approximately one month after the onset of the patient's initial illness, during which time diagnostic studies were carried out, he was found to have a wheeze in the region of the right upper lobe. At this time the roentgenogram had not changed and exploratory thoracotomy was undertaken.

At operation a small bronchogenic carcinoma was found somewhat distal to the origin of the right upper lobe bronchus. This lesion had produced no ulceration, but it had produced a moderate degree of bronchostenosis. There was no evidence for metastatic spread of the lesion to the hilar lymph nodes and the lesion itself was extremely small, measuring less than 2 cm. in diameter. Total right pneumonectomy was performed and all mediastinal nodes were removed. At the time of thoracotomy it appeared that the end-result would be quite favorable in this instance (see Fig. 2b).

Because of the variable behavior of bronchogenic carcinoma, several features of the disease are worthy of comment. The characteristics of behavior which are of great interest are those which produce subtle changes in the thoracic roentgenogram Outstanding are abnormalities of the pulmonary hilum An example seems indicated

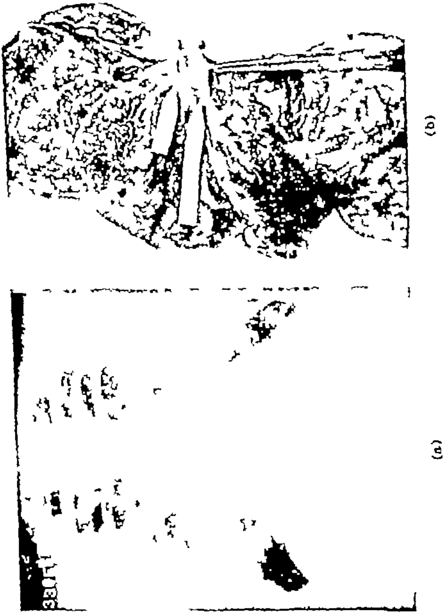

Fig. 2 a—Thoracic roentgenogram of a 63 year old man with a history of cough dyspnea fever and hemoptysis of 10 days' duration. The lesion in the right upper lobe failed to clear and one month after onset of symptoms a wheeze was audible over the right upper lobe b—Small bronchogenic carcinoma in right lung removed at operation. Resected hilar lymph bodies were free of metastases.

Case 3 A 59 year old patient was treated for arteriosclerotic heart disease with angina pectoris for a year A roentgenogram of the thorax had been taken initially This is shown in Fig 3 If one examines it closely one can detect a slight abnormality of the left hilum between the aorta and the pulmonary artery During the period of treatment of his heart disease the patient developed enlargement of a left supraclavicular lymph node. When this node was removed for microscopic examination it was found to contain squamous cell carcinoma considered to be metastatic. It was the opinion of the pathologist that the lesion of the left hilum represented the primary tumor arising in the bronchus.

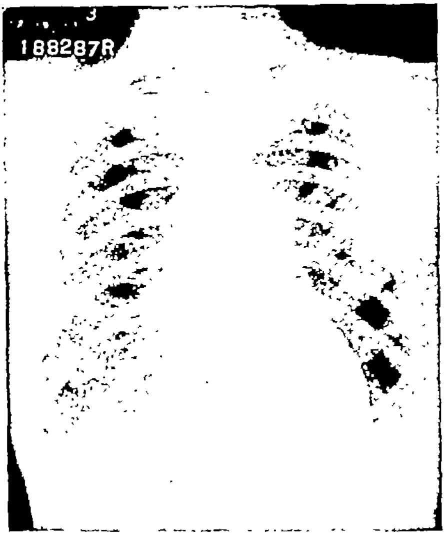

Fig. 3: Thoracic roentgenogram of a 59-year-old man which manifests slight abnormality of the hilar structures in the region between aorta and pulmonary artery. One year after the film shown was taken the patient was discovered to have an enlarged left supraclavicular node. This node was removed for microscopic examination, with the discovery of metastatic squamous cell carcinoma.

This patient exemplifies the fact that very subtle changes in the contour of the hilar shadow should arouse suspicion of bronchogenic carcinoma.

A second subtle change occasionally seen is that of minor shift of the mediastinum, particularly when the cardiac shadow occludes visualization of atelectasis in the basal segments of the left lower lobe.

A third circumstance that should arouse one's suspicion of bronchogenic carcinoma is that of localized emphysema. Fig. 4a shows a film that appears to be within normal limits. It was taken in full inspiration. At *b* in the figure is a film taken in expiration. It demonstrates that the right chest remains inflated during expiration. This of course is diagnostic of obstruction to the main stem bronchus. In this particular instance a bronchogenic carcinoma was discovered in the bronchus to the right lung.

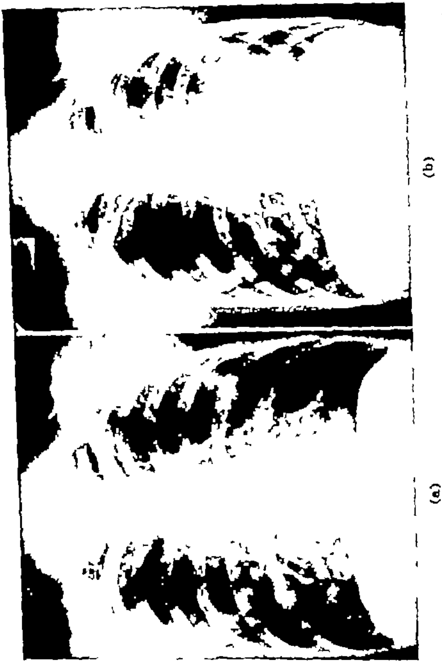
(a) (b)

Fig 1 (a)—Thoracic roentgenogram taken in full inspiration (b)—Thoracic roentgenogram of same patient taken in full expiration. Obstructive emphysema of the right lung is demonstrated. This was discovered to be caused by bronchogenic carcinoma involving the right main stem bronchus

A fourth situation which should arouse one's suspicion is that of the patient with pleural effusion that eludes diagnosis. Peripheral carcinoma of the lung often presents itself symptomatically by the production of pain of pleural origin accompanied by pleural effusion. The traditional diagnostic thought that tuberculosis is a cause of most unexplained pleural effusions must be abandoned. The mere presence of fluid does not mean hopeless pulmonary cancer.

SUMMARY.

In recent years advances in thoracic surgery have made possible extirpative treatment of bronchogenic carcinoma; hence in the light of present knowledge, successful treatment of the patient with bronchogenic carcinoma depends upon early recognition of the disease. This latter can be accomplished only through suspicion on the part of the physician who first sees the patients. An active role can be played by the physician by his insistence upon frequent thoracic roentgenograms for his patients. Unfortunately, there are certain factors which interfere with successful therapy. These consist of the factor of the disease itself, which can be expressed by the statement that bronchogenic carcinoma can progress to an inoperable stage even though the most careful observations are carried out. A second interfering factor is that of the patient himself. This is exemplified by the patient who refuses to follow advice or who ignores obvious symptoms. A third interfering factor lies with the physician himself who fails to lead the patient the full diagnostic route, including surgical exploration of the chest.

There are certain situations in which lie opportunities for the early diagnosis of bronchogenic carcinoma. These consist of the patient with a circumscribed pulmonary lesion, the patient with hemoptysis, the patient with pneumonia which fails to clear, and the patient with a localized wheeze. Certain behavior characteristics of bronchogenic carcinoma which are of diagnostic interest were mentioned. These consist of abnormalities in the hilar area, slight shift of the mediastinum, localized emphysema, and unexplained pleural effusions.

It is emphasized that the responsibility for the early diagnosis of bronchogenic carcinoma lies with the physician who first sees the patient. Suspicion on his part can lead to successful treatment of bronchogenic carcinoma.

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SYMPOSIUM:
BRONCHOGENIC CARCINOMA.
II—RECOGNITION.*

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Benign and malignant tumors produce pulmonary findings characterized for the most part by their location, by the degree of obstruction, and by their sojourn, rather than by their histologic classification. Benign tumors of the bronchial wall, such as polyps, lipomas, papillomas, fibromas and chondromas may give an identical clinical picture to that of a bronchogenic carcinoma; however, in many cases, if there is a minimal degree of complicating bronchopulmonary suppuration, the benign character of the lesion is apparent by the lack of infiltration or destruction of the adjacent bronchial walls.

Bronchial adenomas must be considered as special tumors, exceedingly slow growing, somewhat invasive in character. Their appearance through the bronchoscope is that of a smooth, round mass which is firm, freely bleeding and often covered with a smooth, vascular appearing membrane. Endoscopic removal is sufficient to cure the disease in approximately half of the cases; however, if there is local recurrence, if the tumor bleeds excessively on biopsy, or if there is lung destruction distal to the tumor sufficiently great to necessitate surgical intervention in itsef, surgical removal of the tumor and of the area of lung it has obstructed is indicated.

Closely related to the adenomas, but a distinctly different clinical entity are the cylindromas of the bronchus. These are malignant and, while they, too, are slow growing, they metastasize to the regional lymph nodes and other areas of the body. Therapy here is entirely surgical, although palliative removal of obstructing tissue may be indicated if the position of the lesion indicates it to be nonresectable.

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At present, there are scores of laboratory tests for the determination of the presence of cancer. Few are reliable, and none is sufficiently accurate to suffice without co-ordination with other diagnostic procedures. The most satisfactory results in the cytological diagnosis of pulmonary cancer have been obtained by the examination of material aspirated from bronchi during the course of bronchoscopy. Although collection of bronchial secretions requires bronchoscopy, this is not a real disadvantage inasmuch as all suspected cases of lung cancer should be bronchoscoped.

The location of the site of origin determines whether or not an early lesion may be seen and biopsied bronchoscopically. In advanced cases, the growth may propagate along the bronchial wall and bring it nearer the hilar area. In the great majority of cases in the late stages, the tumor will be visible, and most are accessible for biopsy. In early cases, however, only those that originate in the lobar or first portions of the segmental bronchi will be visible. Telescopic-lens accessories that permit better visualization of segmental bronchi in all lobes are available.

Bronchoscopy serves another purpose: in addition to determining the identity and location if a lesion is visible, it permits evaluation of the status of the airways to the involved lung. If excisional therapy is in the plan, it is essential that everything possible be known about that part of the bronchial system that supplies the sound lung. For diagnosis, bronchoscopy serves in three ways: visualization of the abnormality, provision by biopsy of material for microscopic study, and aspiration of secretions for cytological examination. It must be remembered that in cases of early cancer the majority of examinations will be visually negative.

Bronchogenic carcinoma assumes many variable characteristics. The most common endobronchial form is the soft verrucose, bulky tumor obstructing a major bronchus, or the bronchus to a lobe or a segment. It is red, friable, freely bleeding, and behind it lies atelectatic lung and broncho-pulmonary suppuration in the form of bronchiectasis or lung abscess. The tumor may be covered with exudate, necrotic debris or purulent secretion, which must be aspirated carefully before tissue is taken Other tumors are polypoid in character with a mulberry-like appearance Some carcinomas invade the bronchial walls submucosally, or through the peribronchial lymphatics, to produce an extreme induration of the bronchial walls far above the point of the complete bronchial obstruction, which was determined as the location of the lesion by physical and X-ray findings. Occasionally an ulcerative type of lesion is found with rounded edges and a necrotic base extending along the floor or wall of the bronchus. Such lesions are not often seen bronchoscopically, because at this stage they produce relatively few symptoms which call attention to their presence

Bronchogenic carcinoma also may be found as a small thickening of the bronchial wall with no bronchial obstruction and no pulmonary findings physically or by chest X-rays. Such tumors metastasize extensively and death resulte from the widespread metastases rather than from the primary tumor Only the postmortem examination reveals the apparently insignificant endobronchial lesion

Other bronchogenic carcinomas are collar-button in shape, with a partially obstructing endobronchial lesion and a large extra-bronchial component, which may so compress the bronchus proximal to the intrabronchial tumor that tissue for biopsy cannot be reached

Bronchogenic carcinoma produces no symptoms or findings that may be considered as characteristic of this serious disease, however, it is generally suspected when partial or complete bronchial obstruction is found, or when signs and symptoms of bronchial obstruction recur after having responded temporarily to antibiotic therapy

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The Department of Otolaryngology, University of Illinois College of Medicine, announces its Annual Assembly in Otolaryngology from September 6 to 11, 1954. The entire week will be devoted to surgical anatomy and cadaver dissection of the head and neck, and histopathology of the ear, nose and throat. The Assembly will be under the direction of Maurice F. Snitman, M.D.

Registration will be limited. For information write to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

SYMPOSIUM
BRONCHOGENIC CARCINOMA.
III—TREATMENT* (SURGICAL TREATMENT)

Richard W Yore, M D ,
(By Invitation)
Seattle, Wash

In this brief discussion of certain aspects of lung cancer I would ask your indulgence in any dogmatism that might arise in the course of this portion of the Symposium for the sake of emphasizing and re-emphasizing features of surgical treatment.

Certain general remarks concerning the treatment of pulmonary malignancy might be appropriate and also interesting There has been an alarming and disconcerting wave of pessimism, discouraging both patient and physician, that is not helpful in our continuing efforts in improving the results of treatment. Certainly a healthy degree of realism is both necessary and fair, but an unhealthy deplorable onus of despair has begun to creep into discussions about this disease, on the hospital wards, consultation rooms, and hospital residents' quarters By way of dispelling some of this gloom, one need only remember that the history of the surgical treatment of carcinoma of the lung is just one-half as long as that of surgical therapy of gastric cancer, and yet the over-all results of treatment of these two diseases are the same as measured by the somewhat misleading yardstick—the five-year survival It would be much more fair and reasonable that the surgical treatment of cancer of the lung should be undertaken with an optimistically expectant attitude.

It has been widely accepted that statistically we are facing a serious and ominous real increase in cancer of the lung Dr. Evarts Graham has pointed out that in this country and in England and Wales during the last 25 years, there has been a

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15-fold increase in the incidence of cancer of the lung, whereas in the 15 years between 1930 and 1945 there was an insignificant increase (compared to an increase in the number of people reaching the cancer age) of only 600 deaths from cancer of the stomach. It is not so important, however, that cancer of the lung is more common than gastric cancer, but that every year there is an alarming increased incidence of bronchogenic carcinoma. Dr. Blades, too, in the same discussion, showed that from the Medical Statistics department of the Veterans Administration, where there was a predominant male population of patients, there was a terrifying increase of this disease from 1949 through 1951.

A point that Dr. Morgan has previously mentioned, and which I would like to re-emphasize, is this: no other visceral cancer offers an equal opportunity for detection in the presymptomatic growth period, because of the ease of detecting abnormal areas of density in the normally radiotranslucent pulmonary parenchyma by simple X-ray examination.

Published and unpublished experiences with cancer of the lung have indicated that life expectancy is short following diagnosis, and that the duration of life has not been influenced greatly by any means other than surgical extirpation of the neoplasm; therefore, we should discuss surgical treatment as the primary and most successful form of therapy, and mentions others as ancillary, palliative, or secondary methods.

Surgical extirpation or resection as the treatment of choice in cancer of the lung has three main operative procedures. *(a)* exploratory thoracotomy, *(b)* pneumonectomy, *(c)* lobectomy. These are fairly well standardized operations and have been improved, modified, and made more radical in their scope during the past 15 or 20 years. Gibbon, in a beautifully presented and analyzed series of cases, reviewed also the results of 17 other series reported in the literature and called attention to the fact that there is a direct relationship between the number of cases explored and the number of patients leaving the hospital alive after having had their cancers removed: or, in another way of expression, a doubled rate of exploration yields a doubled rate of resection, and a similar increase in patients leaving the hospital following removal of the malignancy As one examines the various series reported, it becomes apparent that the indications for attempting extirpation are not universally drawn. What, then, might be the indications for thoracotomy? Dr Morgan has already alluded to these by the presentation of the four cases in which his index of suspicion was high, *(1)* solitary circumscribed nodule, *(2)* hemoptysis, *(3)* "pneumonitis," and *(4)* the patient with a localized wheeze Any patient with an undiagnosed, unexplained shadow in the lung, which persists or undergoes slow resolution, and who has all other diagnostic measures negative, should be unhesitatingly explored Even though it is dangerous to point out contraindications to thoracotomy, since they are rapidly changing and disappearing in recent years, we could briefly classify them as absolute and presumptive contraindications

Concerning the procedures of pneumonectomy and lobectomy, it must be pointed out that both of these are performed on the basis of one of two fundamental assumptions, namely, that the disease is a localized process and therefore might be eradicated by surgery, and that removal of the lung or lobes might alleviate suffering arising from pain, hemorrhage, or suppuration in uncontrollable cancer, thus making a limited period of survival more comfortable. The important question of whether significantly better results can be achieved if the whole lung and regional nodes are removed or if only a lobe with its nodes is resected, cannot be answered until a much larger series of lobectomy cases has been thoroughly studied and compared with pneumonectomy results. As it stands today, pneumonectomy is the best operation, providing it entails removal of the entire anatomical unit to include adjacent regional lymph nodes Lobectomy is indicated in the following situations *1* Evidence of diminished pulmonary or cardiac reserve (the danger of producing the "respiratory cripple"), *2* When the carcinoma has extended beyond surgical bounds, but when the bulk of necrotic, suppurative, infective tumor could be removed by lobectomy with hope of relieving the symptoms *3* For profound incapacitating and lingering hemorrhage, for severe pain secondary to chest wall invasion, and for localized abscess and resultant sepsis, even though there are evidences of distant slow-growing metastases

As for "palliative" resections, or resections done in the case of a hopelessly incurable situation, this has been a controversial matter for a number of years. Generally, I believe that removal of necrotic, infected lung tissue reduces the amount of sepsis and continued inflammatory exudate, which the patient would otherwise have to miserably endure. Dr. Ochsner's large series of over 1000 cases showed that 10 per cent of the palliative extirpations survived more than three years, whereas only three of the 200 non-resected cases were alive at the end of two years. Mortality of poor risk palliative resection cases has been reduced from 60 to 20 per cent. This ever decreasing mortality rate, coupled with the personal observation that generally palliative extirpation favorably influences survival time, as well as the patient's ultimate comfort, has influenced many surgeons to recommend and practice this type of surgical approach to a difficult problem.

With an encouraging reduction in operative deaths, which is the result of improved surgical technique, improved anesthesia, blood replacement therapy, and general adoption of antibiotics, there were similarly encouraging advances made in extending the limits of resectability, as by this extension we were able to salvage more patients from the distressing "open and close" category.

Dr. John Gibbon, Jr., made a tremendous contribution in bringing to our attention the technique of intrapericardial ligation of hilar vessels, which increased by 50 per cent the number of successfully resected cases otherwise felt to be non-resectable because of extensive precarious involvement of the large pulmonary arteries, and/or veins. Partial resection of the chest wall, followed by stable reconstruction with tantalum mesh, has increased the number of successfully extirpated peripherally located tumors removed *en bloc* with lobe and segment of chest wall. Similar techniques have been applied to favorable lesions involving a diaphragmatic leaf to still further increase successful radical management of previously considered hopeless cases. It is now commonplace to remove large segments of the pericardium when this layer seems involved by contiguity. In carefully selected cases with an otherwise good prognosis, lesions extending high on one side of the trachea may be "shaved off" beyond microscopic tumor, and the patients survive three to five years comfortably.

Recently, cancers extending to involve the carina have been resected by removal of the lung, resection of the carina, and anastomosis of the contralateral bronchus to the transected trachea by one of several possible methods. Emphasis on techniques of more extensive and complete *en bloc* regional node excision by Cahan, Higginson (of this city) and others, are too recently tried and as yet seldom used in a general way to evaluate their apparent advantage in increasing the survival rates. These workers and their modifications certainly demonstrate a more definitive anatomically organized radical attack on the regional nodes.

In spite of these advancements in techniques, patient care, and improved anaesthetics, the over-all results in treatment of cancer of the lung are quite depressing. In round figures, Dr. De Bakey pointed out that in a given group of 25 cases of pulmonary neoplasm, about 15 of the 25 would meet the criteria of operability and thereby be explored. Approximately nine, or one-third, would prove resectable; about seven patients, or one-fourth of the original number, would survive the operation, and only two, or approximately 8 per cent, would survive five years or longer. To put the situation in even more simple terms, Ochsner stated that out of every three patients with a reasonably certain diagnosis of lung cancer, one would be eliminated because of obviously inoperable growth, one would have non-resectable growth (or possibly refuse surgery), and one would actually have a resectable lesion. In general, in most series a sufficient period of time has not yet elapsed to give an indication of the relationship between the number of patients having had cancer of the lung that leave the hospital following removal of the lesion and the number that survive five years *or more* without evidence of residual cancer. At present there is no indication that there will not be a direct relationship between the two groups. In Gibbon's series most recently published, the analysis covered 532 consecutive proven cases, and the interesting results were as follows: *(a)* 22 per cent operative mortality in patients in whom the cancer was extripated; *(b)* the five-year survival rate was 22 per cent of patients who had cancer removed, and 9 per cent of the entire series. He did not practice palliative resections. In his series an increase in exploration rate of 17 per cent above the next highest rate reported was rewarded by only a 4 per cent increase in the rate of extirpation and a 2 per cent increase in patients leaving the hospital following removal of their cancer.

What are the reasons for such disappointing results in spite of more progressive attacks on the disease? Dr. Morgan mentioned the disease, patient and physician factors responsible for delay or interference with successful therapy. Dr. John Jones in reviewing and analyzing his series of 633 cases brought out that the recent increase in more far-advanced cases is due to increased delay between time of first patient-physician contact and time of surgery recommended. He proposed that increased employment of antibiotics is responsible since they are used to treat recurring lung infections, while the carcinoma remains unsuspected as the cause of the "pneumonitis." In our experience this has been all too true, and notables in this field such as Dr. E. A. Graham and Dr. Robert Janes have preached against incorrect usage of the antibiotics with a low index of suspicion. One other possible reason for patient and/or doctor delay is the false sense of security produced by patient saying to himself "Oh, I can't have cancer of the lung because my chest X-ray a year ago was entirely negative." We must not allow this common misconception concerning chest X-ray findings in early lesion detection.

Just as Dr. Morgan made plea to those of you who do bronchoscopy, so would I like to enlarge upon your importance as a bronchoscopist in the management of suspected cancer of the lung. The bronchoscopist can be of inestimable value to the thoracic surgeon when he provides him with information about fixation, the carina and its configuration and mobility, distortion or concentric narrowing of bronchial lumina as well as bronchial wall biopsy to pick up the submucosal induration of the wall. He should be well and accurately versed in segmental anatomy, not only from the "inside," but also from the "outside"; *i.e.* by X-ray localization, so that "blind" biopsies can be taken from the correct segment involved in the X-ray process. He should develop a strong intellectual curiosity about all and any abnormalities, and bold enough with biopsy forceps to try to confirm microscopically his observation of abnormal findings by a properly gauged biopsy.

In summary, therefore, we have discussed various phases of surgical treatment of carcinoma of the lung, beginning with general remarks about the problem and the alarming increase in incidence, emphasizing the importance of early thoracotomy, mentioning the various forms of surgical treatment and their results. I am sure that it has become most clear to all of you that of all the factors working for the improved results of treatment, the most important is and always will be *suspicion.*

UNIVERSITY OF ILLINOIS COLLEGE OF MEDICINE.

The Department of Otolaryngology, University of Illinois College of Medicine, announces its basic science course in otolaryngology offered by its affiliated hospitals. This combined postgraduate course and residency will begin its 1954-55 session on July 1, 1954. Other openings occur throughout the year. Residencies are available at either the Research and Educational Hospital or the Illinois Eye and Ear Infirmary, or a continuation of the training program may be arranged for the Veterans Administration Hospital at Hines.

A stipend is offered on the following basis: First year residency, $1320 annually; second year residency, $1620 annually; third year residency, $1920 annually.

Application forms are available on request to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12.

OTORHINOLOGIC ASPECTS OF HAND-SCHULLER-CHRISTIAN'S DISEASE.*

J. JULIAN CHISOLM, M.D.,
Baltimore, Md.

Every otolaryngologist should be acquainted with the symptomatology of the clinical syndrome known as Hand-Schüller-Christian's disease. In children it may simulate chronic infections involving the temporal bone and neoplasms or cysts of the paranasal sinuses. It has received far more attention in the pediatric textbooks than in those of our specialty.

Hand, in 1893,[1] described the autopsy findings in a case, but he considered it a peculiar type of tuberculosis. Schüller, in 1915,[2] pointed out the characteristic radiological appearance of the skull defects. Christian, in 1919,[3] published his "Defects in the Membranous Bones Exophthalmos and Diabetes Insipidus" and, thereafter, the name Hand-Schüller-Christian's disease was used to describe cases exhibiting this triad. In 1928, Rowland ascribed the syndrome to a type of xanthomatosis, and for some years thereafter, Hand-Schüller-Christian's syndrome appeared in the pediatric textbooks in the group of disases of abnormal lipid metabolism of obscure origin; however, since 1940,[4] further pathological studies and clinical observations have resulted in the grouping of Hand-Schüller-Christian's disease with two other closely related conditions—Letterer Siwe's disease and eosinophylic granuloma. The three are now generally referred to as variant types of systemic reticuloendotheliosis,[5] the essential change being a proliferative response of embryonal reticular cells of the body to a stimulating and as yet unknown agent. The clinical pictures differ so strikingly that the three are usually classified as separate disease entities, but the pathologic process shows the same basic pattern in each. Goodhill[6] suggests "Histiocytic granuloma" as a better all-inclusive term than systemic reticuloendotheliosis, and reports 18 cases, some of which were

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[*] From the Department of Oto-Laryngology of the Johns Hopkins University School of Medicine.

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followed over a period of years and showed transition from one type to another.

Letterer-Siwe's disease, the acute, rapidly fatal form, usually occurs in infants or before the age of two years. The clinical picture includes general lymphadenopathy, progressive anemia, purpura, fever, skin rash, cachexia and occasional enlargement of the liver and spleen, and skeletal involvement. The lesions are widespread and microscopic examination of them reveals numerous reticular cells, histiocytes, a few giant cells and occasional foam cells, plasma cells and eosinophiles. Siwe suggested that the disease be called "diffuse reticuloendotheliosis." As a rule X-ray therapy does not influence the course of the disease, and death usually occurs within a few weeks or months.

Eosinophilic granuloma, the chronic type of reticuloendotheliosis, was first described by Fraser in 1935.[7] He called it eosinophilic osteomyelitis and considered it a variant of lipoid granulomatosis. The clinical picture is that of a solitary benign tumor of the bone. Under the microscope the tumor is found to be made up chiefly of histiocytes, eosinophiles and giant cells. It may occur in the long bones or the skull and responds equally well to surgical removal or deep X-ray therapy; however, recurrence in other parts of the skeletal system is not uncommon. The same patient may later develop diabetes insipidus or multiple lesions of the Hand-Schüller-Christian type. Love and Fashena[8] reported the case of a child who developed a solitary tumor of the right mandible, diagnosed as eosinophilic granuloma, at the age of 19 months, then three years later diabetes insipidus, and four years later general lymphadenopathy.

The third or subacute type of systemic reticuloendotheliosis is Hand-Schüller-Christian's disease. It has also been called chronic idiopathic xanthomatosis or lipoid granulomatosis. The clinical triad usually associated with it consists of: *1.* X-ray defects in the membranous bones: skull, pelvic girdle and scapulae; *2.* diabetes insipidus; and *3.* exophthalmos. The symptomatology will depend upon the location of the bony lesions. The bones of the orbit and the sella turcica seem to be sites of predilection, which accounts for the frequency of proptosis and diabetes insipidus. When the first symptoms are referable to the temporal bone or the superior maxilla, the case is more likely to fall into the hands of the otolaryngologist at an early stage. He should be on the alert for unusual cases of this type, and X-ray examinations should include the other membranous bones. The films may reveal lesions elsewhere in the skull or in the pelvic girdle or the scapulae, which have as yet given no symptoms. The X-ray changes characteristic of Hand-Schüller-Christian's disease have been likened in appearance to lakes on a map or moth-eaten holes of varying size.

Histologically the lesions are identical in all areas involved and exhibit a peculiar type of inflammatory reaction with eosinophilic and polymorphonuclear leucocytes, a proliferation of reticulum cells, histiocytes, foam cells and giant cells. The foam or xanthoma cells, large mononuclears with lipid vacuoles, are filled with cholesterol. They predominate in the lesions. Their presence accounts for the fact that Hand-Schüller-Christian's disease was originally grouped with xanthomatoses. The proliferative xanthoma-like masses originating in the neighboring bone structures may invade the middle ear cavity, the external auditory canal, the orbit, the antrum, or the pituitary. The characteristic diabetes insipidus is caused by invasion of the pituitary from the sella turcica. In the middle ear, or external auditory canal, the masses may be easily mistaken for aural polypi or cholesteatoma. In the orbit or antrum they may simulate tumors. Goodhill[6] reports the temporal bone findings in a case which came to autopsy and, although there was extensive involvement of the mastoid and petrous pyramid, the bony capsule of the labyrinth resisted invasion, and the cochlea and vestibular apparatus were essentially normal. Eggston and Wolff's textbook of Histopathology[9] pictures a histological section of a temporal bone, which shows the characteristic microscopic appearance. Although the xanthoma cells are rich in cholesterol, the blood level of cholesterol is always normal.

The onset of Hand-Schüller-Christian's disease is insidious and usually between the ages of two and six; however, it sometimes develops at a later age. Troxler and Niemetz[10] report a case in an adult of 35, with diabetes insipidus, generalized xanthomatosis affecting the vertebrae, pelvis and femora, and bilateral lung involvement and pneumothorax. The disease had given its first symptom at the age of 20, when all the teeth began to loosen as the result of changes in the alveolar process. The presenting symptom depends on the location of the first bone lesion—it may be proptosis, diabetes insipidus, a swelling of the cheek suggestive of a malignant tumor or a congenital cyst, a swelling of the mandible with loosening of the lower teeth, or a discharging ear without preceding pain or acute respiratory infection. There may be a low-grade fever, but pain is seldom a symptom. When the lungs are involved there is cough and cyanosis, and X-rays of the chest may reveal changes suggestive of miliary tuberculosis, atelectasis, or cavity formation. Skin lesions are occasionally present and they resemble seborrheic dermatitis, but there is no itching. X-ray changes in the bones must be differentiated from those occurring in multiple myeloma, solitary bone tumors, malignant metastases and chloroma.

The prognosis is uncertain. The bone lesions are usually multiple, tend to resolve spontaneously and to be replaced with fibrous tissue and new bone, only to recur in other areas over a period of years. X-ray therapy causes rapid resolution. The diabetes insipidus can be controlled satisfactorily by pitressin. When secondary infection occurs, as frequently happens in the ear, an appropriate antibiotic is helpful. As the cause or stimulating agent is unknown there is no specific therapy. Death may occur from visceral or brain lesions or a transition to the Letterer-Siwe type; however, after a period of years the condition may become quiescent. If the pituitary has been involved the diabetes insipidus requires continuous treatment, the child's growth is arrested and dwarfism and hypogenitalism become evident as the patient matures. The writer recently had the opportunity of seeing a boy, now 15, who developed Hand-Schüller-Christian's disease at the age of three, the first bony lesion being in the mandible. He then had recurrent involvement of the membranous bones over a period of six years, though no lesions appeared in the temporal bones. He is now an infantile dwarf with diabetes insipidus. It is interesting that while X-rays showed almost complete destruction of the sella turcica, in the early stages of the disease, it has now regenerated. Recent films reveal a well formed but curiously shaped sella with elongated clinoid processes.

CASE REPORTS.

Case 1. G. H., white, age 27 months, first seen by me June 22, 1949. Referred by Dr. Harriet Guild of the Pediatric Department, for a routine check-up.

Personal History: No ear infections. In the summer of 1948 he developed a tumor of the left orbit—X-rays in the Pediatric Department August 1, 1948, resulted in a diagnosis of Hand-Schüller-Christian's disease. The proptosis disappeared completely after X-ray therapy, and there has been no recurrence.

Examination: June 22, 1949, revealed no abnormality in the ears, nose and throat. X-rays of the membranous bones revealed no new lesions. He was next seen for a check-up November 4, 1953, then six and one-half years old and large for his age. In the spring of 1951, he had developed a lesion in the pelvic bone, which cleared up with X-ray therapy; otherwise, there have been no further manifestations of Hand-Schüller Christian's disease.

At this time, he had a cough due to infection of the adenoids. The right antrum was rather dark on transillumination. X-ray of the sinuses revealed some marginal clouding of the right antrum, but no bone lesions in the superior maxilla. X-rays of the skull and pelvis were negative. This case illustrates a mild type of the syndrome with only two bone lesions: one in the skull, and one in the pelvis, over a period of more than five years. He has not had diabetes insipidus.

Case 2. J. F., female, age three and one-half years. First seen by me June 30, 1951. Referred by Dr. Harriet Guild of the Pediatric Department.

Complaint: Discharge from the left ear, impairment of hearing.

Past History: Bilateral otitis media at 18 months with several recurrences since then. About February 1, 1951, mother noticed a soft painless swelling in the scalp of the occiput just above the hairline, which was thought to be a cyst under the skin.

Present Illness: On March 1 mother took the child to her local pediatrician because she was very irritable, though there were no specific complaints. He noted a swelling in the roof of the left external auditory canal, suggestive of a localized infection in the skin. Several weeks later, a polyp appeared in the left canal, and it was removed by an otologist. A second polyp developed within a few weeks, and it was removed under general anesthesia May 24, 1951. After this the left ear began to discharge, and the hearing in both ears seemed impaired.

Examination: June 30, 1951. Tonsils and adenoids enlarged. *Ears:* right drum intact, opaque; left canal filled with foul smelling purulent discharge and a large, pale yellowish, polypoid mass arising from the roof of the bony canal; the left drum could not be seen. There was obvious impairment for test words in both ears. The skin over the left mastoid and the left post-auricular fold exhibited eczematous changes. There was a discharging sinus tract in the occipital region.

Culture: left ear: staphylococcus albus, resistant to penicillin, sensitive to aureomycin and terramycin. X-rays taken June 29, 1951, showed a very large area of destruction in the occipital bone and multiple areas in both petrous pyramids (see Fig. 1). There was also extensive destruction in the left temporal bone.

Fig. 1 (Case 2). X-ray of base of skull, taken June 28, 1951, showing large area of destruction in the occipital bone and multiple areas in both petrous pyramids.

The child was started on aureomycin and a course of X-ray therapy. She returned July 30, 1951, after having had nine X-ray treatments over a period of three weeks. The left ear was clean and dry, the drum intact, the polyp had completely disappeared and a small defect could be seen in the roof of the bony canal. The discharging sinus tract in the occiput had healed. Repeat X-ray films on July 31, 1951, showed no change as yet in the appearance of the left temporal and occipital bones (see Fig. 2). No other lesions had appeared. Since then, the child has been brought back to Baltimore repeatedly, and a succession of other bone lesions have developed and then regressed under X-ray therapy.

A brief resume of events follows: December 17, 1951. *Left ear:* normal, defect in bony canal healed. *Right ear:* skin over the mastoid

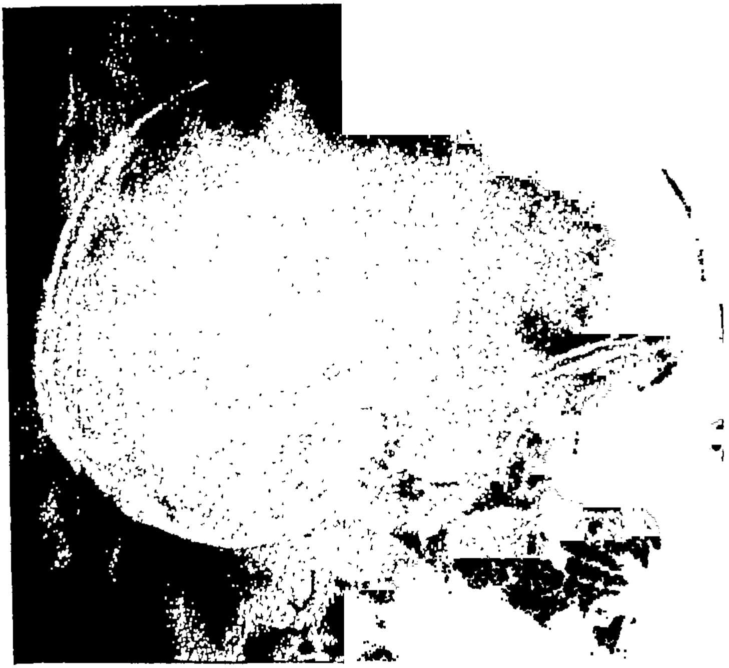

Fig. 2 (Case 2). Lateral X-ray of skull taken July 31, 1951, showing extensive areas of destruction in the left temporal and occipital bones.

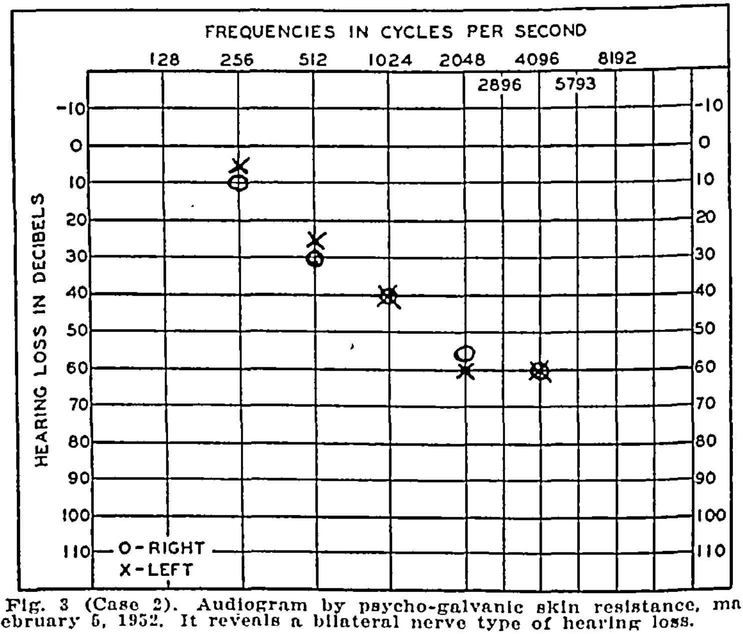

Fig. 3 (Case 2). Audiogram by psycho-galvanic skin resistance, made February 5, 1952. It reveals a bilateral nerve type of hearing loss.

eczematous; large cholesteatoma-like mass filling the canal and arising from the roof. It was easily removed with a small curette, had a foul odor, and a culture made from it was reported staphylococcus albus At its site of attachment, there was a bony defect in the roof of the canal, filled with pale granulation tissue. X-rays revealed changes in the right temporal bone similar to those in the left, described above. A new lesion has appeared in the occipital bone and the child has just developed diabetes insipidus

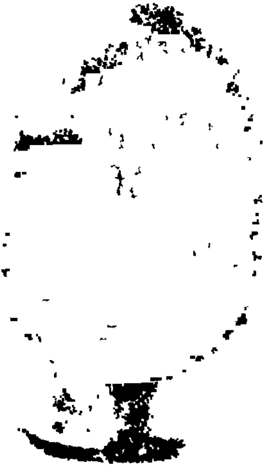

Fig 4 (Case 2) Anterior-posterior view of skull made May 29 1952, showing marked clouding of the right maxillary sinus

Another course of X-ray therapy was started February 5, 1952 Both canals look normal, right drum opaque and thickened, left drum normal Psychogalvanic skin resistance hearing tests verify a subjective audiogram done October 11, 1951 There is a marked bilateral impairment of hearing of the nerve type (see Fig 3) Diabetes insipidus well controlled with pitressin New lesions have appeared in the pelvic bones

May 22, 1952: Ears look normal New lesions have appeared in the skull The face looks normal, but anterior-posterior X-rays of the skull taken May 29, 1952, show marked clouding of the right antrum (see Fig. 4) November 13, 1952: Swelling of the right cheek first noted two weeks ago Nose looks normal. There is a definite protrusion of the right eye and a firm swelling of the right cheek, which might easily be mistaken for a malignant tumor of the right antrum.

February 13, 1953: Swelling of the right cheek subsided. New lesions in the occiput and mandible. April 10, 1953: X-rays of the skull show extent of repair of bone changes seen in Fig. 2 (see Fig. 5). October 14, 1953: Audiogram (subjective) made today shows a drop of 10 db in both ears for C256, a drop of 20 db for C4096 in the right ear and an apparent improvement of 30 db for C4096 in the left ear.

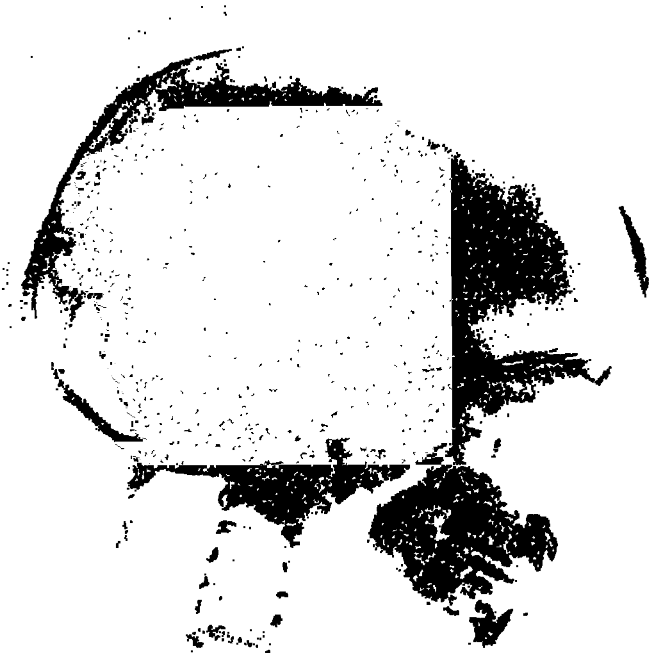

Fig. 5 (Case 2). Lateral X-rays of skull taken April 10, 1953, showing extent of repair of bone changes seen in Fig. 2. There is also a new area in the left parietal bone.

The proptosis of the right eye recurred in the spring, began to regress August 20, 1953, and has varied in degree since then. X-rays show a defect in the supra-orbital ridge on the right and a new lesion in the posterior wall of the left orbit. Lesions in the right parietal region and the left pubis are healing.

Case 3: M. L., female, age nine years. Admitted to the Johns Hopkins Hospital July 27, 1953, as a patient of Dr. Walter E. Loch. *Complaint:* Impairment of hearing.

Family History: Allergy on both sides. *Past History:* Infantile eczema. Asthma since six months of age. No trouble with ears until March, 1950, when at the age of five years nine months, she developed purulent discharge from the left ear, accompanied by post-auricular swelling.

The left mastoid was opened, and a month later the wound healed. In June, 1950, after swimming, both ears began to discharge, hearing became impaired and she complained of dizziness. In August, 1950, a bilateral mastoidectomy was done. Both ears became dry, but the hearing remained poor. In June, 1952, she developed diabetes insipidus. In December, 1952, both ears began to drain again and the discharge from the left ear persisted in spite of antibiotic therapy.

She was brought to Baltimore for the first time in July, 1953. Examination on admission July 27, 1953. Upper respiratory tract normal. Left ear: draining mastoid fistula, blood tinged purulent discharge in the external canal and a mass adherent to the anterior wall in the mid-portion, hiding the drum. Hearing: Left ear nil; right ear profound loss. X-rays showed defects in both petrous bones, no demonstrable lesions elsewhere in the skull and a questionable lesion in the right pubis.

On the basis of the history of diabetes insipidus, Hand-Schüller-Christian's disease was suspected, in spite of the absence of characteristic X-ray findings. Radical mastoidectomy, left, July 30, 1953 (Dr. Loch). The cavity was filled with grayish-yellow tissue (frozen section diagnosed acute inflammatory reaction). The lateral sinus and the dura of the posterior and middle fossae were widely exposed and covered with granulations. A large labyrinth sequestrum was removed, exposing the internal carotid artery. The middle ear was filled with granulations and yellowish tissue; no ossicles could be demonstrated. The operator noted that the gross appearance confirmed the suspicion of Hand-Schüller-Christian's disease.

Pathological report (labyrinth sequestrum): Acute and chronic inflammatory reaction with many eosinophiles, large mononuclear cells and necrosis, marrow spaces infiltrated with small mononuclears, eosinophiles and giant cells. Diagnosis: Hand-Schüller-Christian's disease. X-ray therapy was begun August 10, 1953. Patient was last seen Novembers 29, 1953. Ear dry. X-rays show continued healing of defects in the petrous bones and no new bone lesions elsewhere. Has gained nine pounds in weight.

CONCLUSIONS.

1. Hand-Schüller-Christian's disease may simulate middle ear or mastoid suppuration in children. The sudden appearance of an aural polyp in a previously normal ear should make one suspicious. X-ray examinations of suspected cases should include the entire skull and other membranous bones, as well as the temporal bones. A history of diabetes insipidus is a valuable clue.

2. If the case is diagnosed early, X-ray therapy plus an appropriate antibiotic can result in rapid resolution (Case 2); however, multiple lesions in the petrous pyramids may lead to a permanent hearing loss of the nerve type, probably due to encroachment on the internal auditory canal (see audiogram, Fig. 3). Autopsy reports reveal that the capsule of the cochlea is resistant to invasion, yet in Case 3, repeated and undiagnosed lesions in the temporal bone resulted in a sequestration of the labyrinth and a total loss of hearing.

3. Hand-Schüller-Christian's disease may simulate tumors of the antrum as well as orbital tumors. X-ray of the sinuses alone may fail to reveal the true nature of the disease.

4. The prognosis is uncertain. Although the bone lesions respond well to X-ray therapy, they tend to recur in all the membranous bones over a period of years. Case 2 has had innumerable lesions in the past three years. Cases with pituitary involvement and diabetes insipidus, if they survive the early years, are likely to be infantile dwarfs.

5. The bone lesions tend to resolve so completely that X-rays several months or years later may reveal little or no trace of them; thus, in a case of diabetes insipidus, due originally to destructive lesions in the sella turcica, X-ray films after several years may picture a fairly normal sella.

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FURTHER EXPERIENCES WITH DIHYDROGENATED ERGOT ALKALOIDS IN CERTAIN OTOLARYNGOLOGIC DISORDERS.

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and
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We recently reported the results obtained in the treatment of 116 cases, with sublingual Hydergine (R), a sympathetic inhibitor.[1] Since the preliminary report we have continued using this mixture of dihydrogenated ergot alkaloids, and our purpose in this presentation is to report our results in an additional 210 cases (see Table I).

TABLE I—TYPES OF CASES.

Vertigo	77
Hypertensive epistaxis	10
Dryness of the mouth	30
Glossodynia	19
Post-nasal discharge	[illegible]
Atrophic rhinitis and nasopharyngitis	17
Miscellaneous	5
Total this series	210
Previous series	116
Total clinical evaluation	[illegible]

Just as was emphasized in our original communication, we are aware that the clinical behavior of many of the disorders treated is erratic, and spontaneous regression may be as sudden and complete as it was in many of the cases we report. In order to eliminate this pitfall, we are at present undertaking a blind placebo study at Charity Hospital in New Orleans. Our intention is to publish the results of this clinical evaluation at a later date.

PHARMACOLOGY.

The best-known action of the ergotamine and ergotoxine drugs is vasoconstriction; not so well-known is the fact that they also have a sympathetic blocking action. Recently,[2] as a

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result of hydrogenation of the ergotoxine group, it has been possible to eliminate the vasocontrictor action so that the sympathetic blocking action becomes predominant.

Other actions ascribed to Hydergine are:

1. Depression of the central vasomotor centers with resultant vasodilatation and in some cases, a fall in blood pressure.[3]

2. Peripheral vasodilatation.[4]

3. Central inhibition of pressor-receptor reflexes which bring about compensatory vasocontriction and arterial blood pressure rise.[5]

4. Centrally induced bradycardia; this is rather unusual among sympathetic blocking agents.[6]

5. Central sedative action; also augmentation of barbiturate action.[7]

6. An adrenosympathicolytic effect.[8]

7. A possibility that oxygen utilization might be increased.[9]

8. Decrease in vascular distensibility, increase in venous pressure and increasing prominence of arterial pulsations.[10]

Toxic Manifestations. In this portion of our study, only two individuals reported untoward reactions: both were minor and subsided promptly when the drug was discontinued.

RATIONALE OF TREATMENT WITH HYDERGINE.

Vertigo. The most widely-held present-day concept of the pathologic physiology of many cases of labyrinthine vertigo is that there is arteriolar spasm with dilatation of the capillary-venule network distal to the spastic segment. It is felt that in the distended loop there is sludging of blood, liberation of toxic metabolites, among which is histamine; anoxia of the blood vessel walls, and increased capillary permeability which leads to tissue edema and possibly increased accumulation of endolymph.

The effectiveness of Hydergine in these cases may be the result of a combination of its pharmacodynamic actions. The vascular spasm is probably relieved by the peripheral vasodilatation. Once relieved, the spastic effect is probably prevented from recurring by depression of the central vasomotor center; further, the central sedative effect undoubtedly serves to allay some of the psychosomatic element usually present in these conditions. Other phenomena of importance may be the fact that during Hydergine therapy a decrease in vascular distensibility, increase in venous pressure, and increasing prominence of arterial pulsations have been noted; this would undoubtedly serve to relieve the congested state of the part. Finally if, indeed, Hydergine does increase oxygen absorption or utilization, it may help to reestablish enzymatic and other chemical homeostasis in the affected tissues.

Epistaxis of Hypertensive Origin. In the management of epistaxis of hypertensive origin, more than merely stopping the bleeding is desirable; attempts to lower the blood pressure should also be made.

Most cases of hypertension are now believed to be of psychosomatic origin; those which are not, undoubtedly have developed some psychosomatic overlay, this being particularly true in the hypertensive epistaxis. The psychosomatic disturbance is said to be mediated through the autonomic nervous system in the form of relative sympathetic preponderance, with resultant generalized vasconstriction of the vascular bed. In the kidneys, vasconstriction causes elaboration of long-acting pressor substances into the circulation.

The use of Hydergine in these cases is predicated on its ability to depress the sympathetic neurogenic influences, to provide peripheral vasodilatation, to provide central sedation (in addition to augmenting barbiturate action), and to lower the blood pressure; all these effects are obtained with the production of none of the signs of vasomotor collapse.

Dryness of the Mouth. Salivary glands have both a sympathetic and parasympathetic nerve supply. They differ from other effector organs, however, in that both elements of the autonomic nervous system are cholinergic.[11] A decrease in salivary output, therefore, could be due either to a hypofunctioning autonomic nervous system or to hypofunctioning acini of the glands (possible, in some instances, at least, on the basis of a decreased blood supply). The effectiveness of Hydergine may be due to its peripheral vasodilator action, or to the depression of the central vasomotor system. Most cases respond to Hydergine alone, but it may be necessary to use prostigmin bromide in addition, for its action in destroying choline esterase and thus permitting freer transmission of cholinergic impulses.

Parenthetically, we might mention that we have used hydergine in a number of cases complaining of the "dry, scratchy feeling" in the nose, nasopharynx, pharynx, or larynx that often is the harbinger of the common cold or a catarrhal upper respiratory infection. Our reasoning was that if the mucous glands of the area could be stimulated, the condition would be relieved; this proved to be the case in many instances.

Glossodynia. Hydergine was used in patients complaining of painful, burning tongue, on the theory that, in some cases at least, the symptoms might be the result of a circulatory disturbance and, therefore, would be benefited by vasodilatation, the increased blood flow leading to improved nutrition and a "flushing out" of accumulated toxic metabolites. It is interesting to note that some of the vitamins used in the past are potent vasodilators.

POSTNASAL DISCHARGE.

Patients complain of nonpurulent postnasal discharge either *1.* when the amount of mucus is profuse, *e.g.*, as in allergic rhinitis, or *2.* when the mucus is thick and does not move normally, or rolls over in masses or globules. In the latter instance the individual becomes conscious of the thickened mucus and begins to make strenuous efforts to remove it, sometimes going so far as to produce nausea and vomiting.

Our rationale in trying Hydergine in these cases was based upon the hope that blockage of the sympathetic nerve supply would leave the parasympathetic secretory system unopposed, and secondly, that the vasodilatation produced would improve the blood supply and thus re-establish normal viscosity. The object was not to "cure," or abolish, the postnasal discharge; rather, it was to re-establish the normal mucus pattern.

ATROPHIC RHINITIS AND NASOPHARYNGITIS.

There is general agreement that in ozena there is a decrease in the blood supply to the nasal mucosa. Whether this is primary or secondary to other factors is not known; nevertheless, there have been reports concerning the usefulness of vasodilating agents in this condition. It was for this reason that Hydergine was used in these cases.

MISCELLANEOUS CONDITIONS.

We have used Hydergine in several cases of Bell's palsy in an attempt to restore circulatory dynamics in the facial nerve, as it courses through the Fallopian canal of the temporal bone. The rationale of its use here is the same as that for the use of histamine in this condition.

Several cases of cranial nerve neuralgia were administered Hydergine, because it has been suggested that these conditions may be due to ischemia of the nuclei.

DOSAGE.

The medication was administered in the form of sublingual tablets containing equal parts of the three hydrogenated alkaloids of ergotoxine.* The patients were instructed to dissolve one or more tablets under the tongue four to six times daily.

RESULTS.

Vertigo. Sixty-four of 77 cases demonstrated marked to complete remission of symptoms; the remainder either reported no relief or only slight improvement.

Hypertensive Epistaxis. It was our clinical impression that the use of Hydergine in these 11 cases made their management much simpler. Objectively, the decrease in blood pressure was usually dramatic and similar to that recounted in our previous report.

Dryness of the Mouth. The results in this condition were singularly impressive; 27 of the 30 patients reported marked improvement to complete remission; two patients required a combination of Hydergine and prostigmin bromide to obtain complete remission. In one instance the treatment was completely unsuccessful.

Glossodynia. Of the 19 patients with this complaint, 15 reported rapid relief of symptoms, two were moderately improved, and two noted no improvement.

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* The Hydergine (P) used in this study was provided by the Sandoz Pharmaceutical Co.

Postnasal Discharge. All but five of the 58 patients who presented themselves with a thick, tenacious, mucoid postnasal discharge reported marked improvement following a course of Hydergine. Most of these individuals displayed a mild anxiety state, or smoked a considerable number of cigarettes each day.

Atrophic Rhinitis and Nasopharyngitis. Thirteen of the 17 cases with this complaint demonstrated improvement, but relapsed to their former state when the medication was discontinued. There was no noticeable improvement in four cases.

Bell's Palsy. Six patients with unilateral Bell's palsy were treated with Hydergine. Though difficult to evaluate clinically, it was our impression that resolution was speeded.

Cranial Nerve Neuralgia. Three patients with cranial nerve neuralgias were included in the series; two patients showed no improvement. In one individual the attacks seemed to be of a milder nature. This may have been coincidental.

SUMMARY.

1. A report is made of further results of a clinical evaluation of the use of a new adrenergic blocking agent, Hydergine, in several otolaryngologic disorders. Included among these disorders are: vertigo, hypertensive epistaxis, dryness of the mouth, glossodynia, postnasal discharge, atrophic rhinitis and nasopharyngitis, and Bell's palsy.

2. The pharmacology of the dihydrogenated ergot alkaloids is again reviewed.

3. The rationale for the use of hydergine in the disorders considered is presented.

4. The results obtained roughly approximate those of the first clinical evaluation. This leads us to conclude that Hydergine is a useful addition to our therapeutic armamentarium.

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705 Baronne Building.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1 and continue for a period of ten months. Registrations must be completed before August 15 Detailed information and application forms may be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minnesota

SURGICAL TREATMENT OF THE FRONTAL SINUS BY THE EXTERNAL ROUTE.

J. M. TATO, M.D.; D. W. SIBBALD, M.D.,
and
O. E. BERGAGLIO, M. D.,
Buenos Aires, Argentina.

1. A study of the surgical treatment of chronic frontal sinusitis shows early two fundamental and divergent points of view, one directed to the obliteration of the sinus and the other to its conservation with improved intranasal drainage.

The earliest forms of surgery were reserved for the cure of necrosis of the walls and of fistulas.

Runge[1] of Germany, conceived the idea of obliterating the sinus cavity by opening or removing the anterior wall, and treating the tissues with a caustic ointment of nitrate of silver, in an endeavor to unite the soft tissues and mucous membrane lining the bone. Thus, was initiated the idea of the elimination of the sinus cavity.

The majority of surgeons, however, have devoted themselves to the establishment of an efficient and permanent communication between the sinus and the nose.

This paper is based on the conception of the obliteration of the sinus and the closing of the fronto-nasal duct.

This concept is shared with R. R. Woods[2] of Dublin, whose whose opinion is quoted, "Teaching has made us all believe that to succeed in frontal sinus operations, a patent frontal duct must be maintained. I am suggesting that the teaching is quite wrong. For success in this operation the frontal duct must be closed."

2. In general, surgical treatment by the external route is indicated in the following cases:

1. Simple or complicated infections in which medical or surgical treatment has failed to cure or improve the patient's condition.

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Simple cases can be divided into those showing

a. Insufficient natural drainage.
b. Irreversible changes in the mucous membrane.
c. Pyocele.

Complicated cases may be divided into those of

d. Intracraneal complications.
e. Cranial, osteomyelitis of the vault.
f. Orbital.
g. General.

2. Mucocele.

3. Osteomas and other rare tumors of pseudotumoral granulomas.

4. Sinus troubles due to irreversible allergic conditions which block the fronto-ethmoidal infundibulum, provoking pain which does not cede to ordinary medical treatment.

5. As a means of intracranial access to the roof of the ethmoid and the cribriform plate (olfactory fossa) in cases of fissure of the ethmoid with a resultant cranial hydrorrhea.

6. Pneumocraneum of frontal sinus origin.

7. Trauma of the frontal sinus.

Speaking in general terms of the infections of the frontal sinus (which form the majority of the surgical cases), the following procedures may be cited as classical.

1. Ethmoidectomy with removal of the fronto-ethmoidal wall.

2. Opening of the frontal sinus by resection of the

a. Inferior wall. Jansen, Ritter.[3]

b. Partial removal of the inferior wall. Uffenorde-Seiffert-Lynch.[4]

c. Partial removal of the frontal wall. Ogston.[5]

d. Entire removal of the anterior wall. Kuhnt.[6]

e. Total removal of the inferior wall and part of the anterior (Killian-Taptas[7]), etc.

f. Total removal of the inferior wall and partial removal of the anterior, but respecting the integrity of the ostium of the frontal sinus (Walsh).

g. Total removal of the anterior and inferior walls (Riedel[8]).

h. Removal of anterior and posterior walls of the frontal sinus leaving the inferior wall and the fronto-nasal duct or else packing the latter with gelatin sponge soaked in fibrin. Opening of the anterior ethmoidal cells or exceptionally the posterior. The sinus is packed postoperatively with gauze to encourage granulations from the dura and bone to bring about gradual obliteration of the cavity (Mosher[9]).

i. R. R. Woods[10] of Dublin takes away the inferior wall of the frontal sinus and meticulously removes the mucous membrane of the cavity and of the fronto-nasal duct, achieving eventual obliteration of the sinus and duct.

j. Finally we come to the osteo-plastic technique or temporary opening of the anterior wall which was first practiced in 1894 by Brieger and Schönborn,[11] modified by Bergara and more recently by Tato of Buenos Aires.

All other procedures represent modifications of the foregoing list. In the United States the Lynch operation and in England the Howarth operation have many followers.

3. Treatment through the surgical opening of the mucous membrane of the sinus.

a. Total extirpation.

b. Partial extirpation.

c. Conservation of the mucous membrane.

4. Establishment of a permanent fronto-ethmo-nasal communication by

a. Simple enlargement.

b. Flaps using nasal mucous membrane.

c. Free grafts of skin.

d. Tubes—Rubber, Acrylic, Tantalum.

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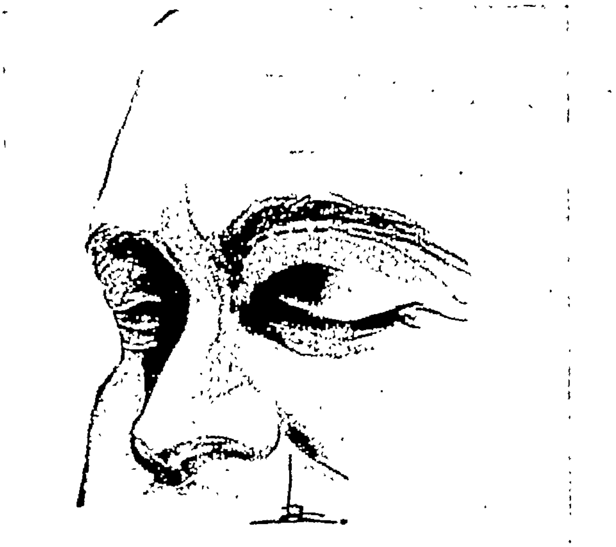
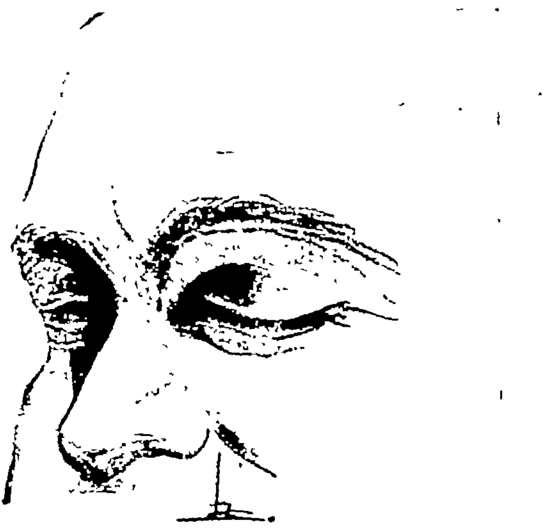


FIG. 2.

the anterior wall by formation of an osteo-plastic flap is the procedure of choice. In cases of bilateral sinusitis, both sinuses may be dealt with from one side by complete removal of the intersinal wall (see Fig. 12); however, in such cases we do a total removal of the inferior wall and the antero-internal portion of the anterior wall below the esthetic eminence.

b. Complete and meticulous removal of the mucous membrane of the sinus, respecting that of the infundibulum.

2. Closure of the communication with the nose:—This is done by separating the mucous membrane from the canal wall sufficiently to form a cuff which is then inverted downwards (see Fig. 9). In case of doubt about closure, a piece of fascia lata or a flap of periosteum from the neighboring bone may be inserted with fat placed above it (see Fig. 10). If the fatty tissue has some aponeurosis attached to it, this latter may be introduced into the infundibulum and suffices to close the opening without anything further being done.



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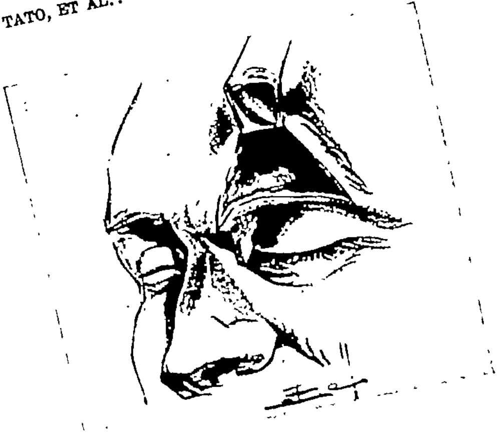

Fig. 3.

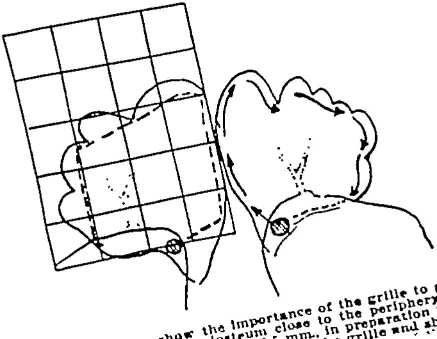

Fig. 4. A diagram to show the importance of the grille to guide the
geon in the section of the periosteum close to the periphery of the sir.
exposing the bare bone for a width of 5 mm. in preparation for the [illegible]
burr, which follows the path mapped out by the grille and shown by the
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burr inferiorly. The [illegible] e of

3. We do an ethmoidectomy if it is necessary, practicing the transmaxillary route operation (Ermiro de Lima). We always respect the fronto-ethmoidal wall, which should remain intact.

At this point we may consider the question of the removal of the ethmoid. Woods[12] states in the description of his technique that on the whole a wide removal of the ethmoid is not usually practiced; but if any ethmoidal cells are opened, the floor of the frontal sinus is conserved, to avoid making a large opening into the nose, which later would be difficult to close.

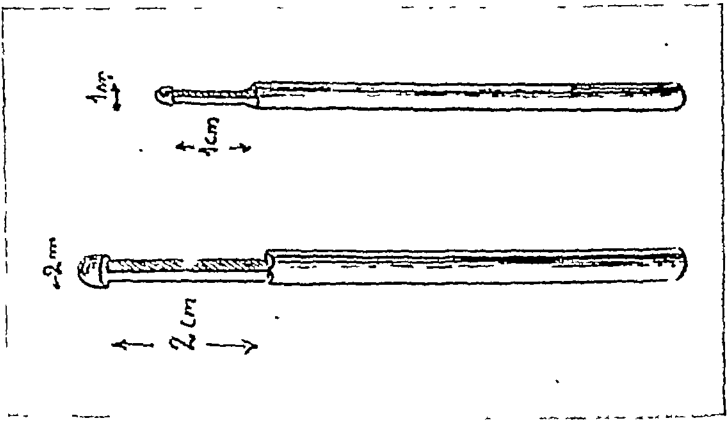

Fig. 5. Burr. Tato's model with smooth spherical head and cutting laterally.

He finally adds that his experience does not support the statement that the ethmoid is the key to the frontal sinus.

In this attitude, he has the approval of Mosher[13] who states that in cases of osteomyelitis of the frontal bone and sinus, he often left the infected ethmoidal cells for a further operaon and later found this was not necessary. It would appear hat surgeons have been putting the cart before the horse and the logical deduction to be drawn is that the frontal sinus infects the ethmoidal cells, which is contrary to the convictions of many of us.

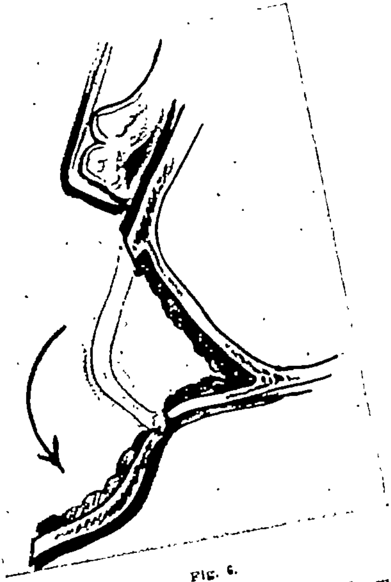

FIG. 6.

In the presence of mixed pansinusitis (allergy plus infection) characterized by a polypoid degeneration of the ethmoid and maxillary sinuses and to a lesser degree in the sphenoidal, and rarer still, in the frontal, Tato has found that the Ermiro de Lima[11] technique (transmaxillary ethmoido-sphenoidectomy) gives satisfactory results in the great majority of

4. Now comes the original note in the operation, the filling of the cavity with fat or other material. When the osteo-periosteal technique is necessary, in large and high sinuses, the procedure Tato outlines is as follows:

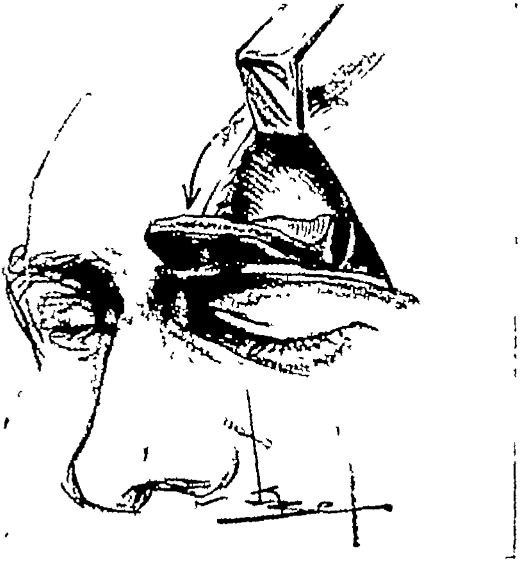

Fig. 7.

a. Radiography of the sinus is made with the addition of a wire grille with square of 1 cm. which is applied to the frontal bone (see Fig. 1) and the head is placed in the fronto-nasal position. Lately we have been following the current practice of a Buenos Aires surgeon, Oliveri, who carefully cuts out the contour of the sinus from the X-ray film, and after the skin has been raised applies it *in situ* over the sinus, so as to enable the surgeon to trace accurately his incision of the periosteum. To achieve the same end, Ermiro de Lima introduces a bronchoscopic lamp into the sinus through a trephine opening. These practices enable one to establish the exact dimensions of the frontal sinus, with a view to future surgery.

b. Anesthesia: Local with pre-anesthesia and, exceptionally, a general anesthetic.

c. Incision of the skin and subcutaneous tissues following the inferior margin of the eyebrow as generally practiced, but extending somewhat laterally (see Fig. 2). The periosteum is not incised. It must be preserved untouched.

d. Separation of the skin and subcutaneous tissue upwards and exposure of the area corresponding to the anterior wall of the sinus with its adherent periosteum and then extending somewhat beyond the sinus limits (see Fig. 3).

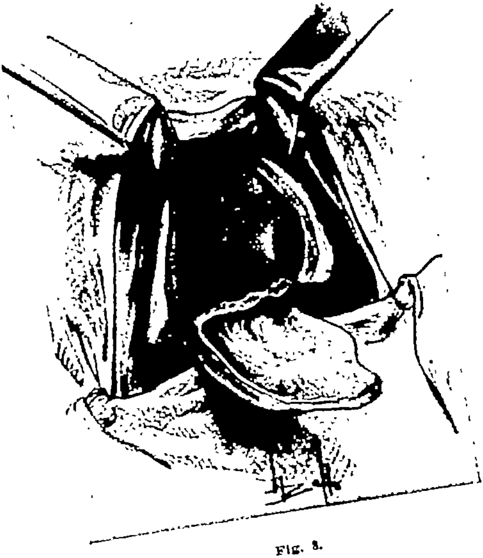

Fig. 3.

e. The periosteum is incised in accordance with the contour of the sinus by studying the grille radiograph (see Fig. 4), with its centimeter squares, or following Oliveri's or de Lima's methods. By these means a strip of bare bone of 5 mm. width is traced and forms the guide for the future use of the special burr.

f. To enter the sinus please take note of the classical landmark, the nasal process of the frontal bone and perforate it immediately above the lachrymal portion of the os unguis (see Fig. 4). We use a common perforating burr to make the initial step.

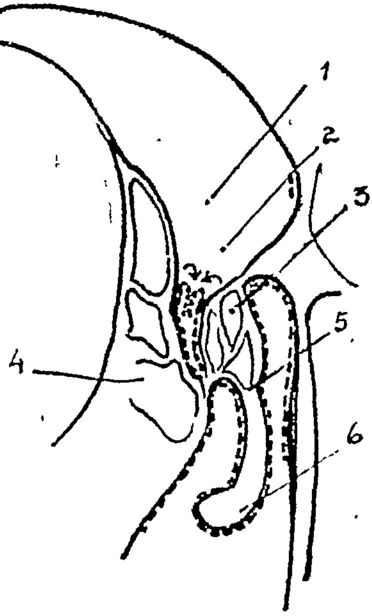

Fig. 9.

1. Frontal sinus, peri-infundibular area.
2. Ostium of the frontal sinus The arrows indicate the invagination of the peri-infundibular mucous membrane, which is marked by a dotted line.
3. Peri-infundibular cells
4. Anterior ethmoidal cells
5. Insertion of middle turbinate.
6. Free portion of middle turbinate.

g. The most useful burr is one especially designed by Tato for frontal sinus work (see Fig. 5). It has three cutting edges and a smooth spherical head, which avoids a possible perforation of the posterior wall of the sinus and is known as a reamer. It is made by the Storz Instrument Co., of St. Louis. Continuous lavage with normal saline and suction is used during this stage of the operation. The burr is inclined obliquely, so as to obtain a bevelled edge. Starting from the initial perforation, the reamer follows the contour of the sinus, which has previously been established by accurate measurements from the grille radiograph and subsequently traced. The course of the reamer is in and up, and out and down.

h. With a chisel as a lever, the osteoplastic flap is turned downward by fracturing the bone just below the superciliary ridge, the periosteum being left intact. In this way an excellent view of the interior of the sinus is obtained (see Figs. 6, 7, and 8).

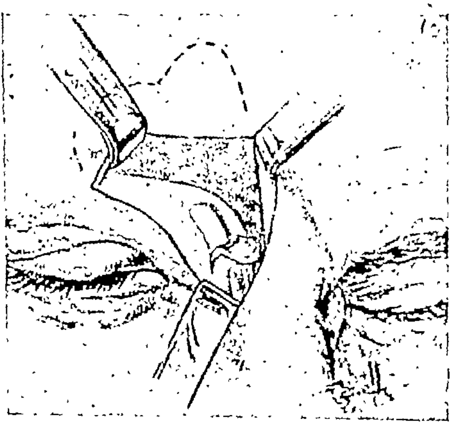

FIG. 10.

i. The mucous membrane of the frontal sinus and that of the interfrontal septum is removed with meticulous care. We use for this purpose a Freer's septal elevator and in addition a spherical cutting burr or diamond burr of appropriate diameter which is very efficient in narrow hidden recesses. The burr is the best curette. We also eliminate all the intra frontal septa, smoothing them down.

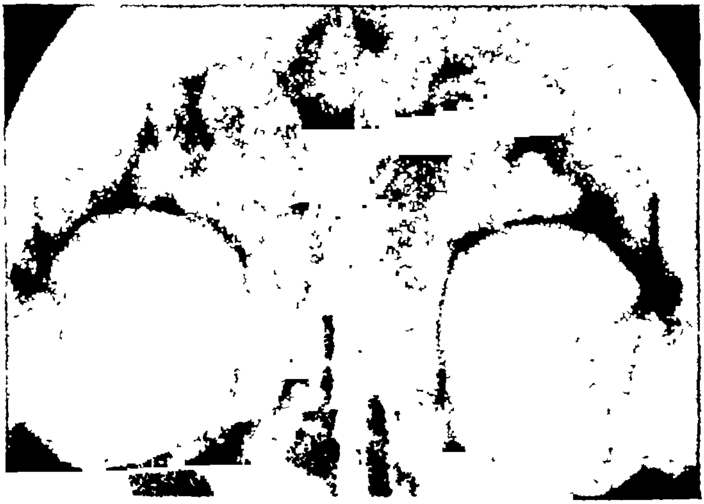

Fig. 11. Pre-operative radiograph.

j. The mucous membrane of the infundibulum is separated from the contour of the duct, invaginated, and pushed downwards to secure permanent closure of the duct (see Fig. 9).

If any doubt exists about a perfect occlusion, a piece of fascia lata attached to the fat is inserted into the opening and left there, or a flap of periosteum from the neighboring bone is inserted as Bergaglio and Alba have done.

k. The cavity is filled with fatty tissue from the abdomen or thigh, and if possible, with the corresponding fibrous tissue sheath attached. We take enough for our purpose. Actually we are now using fibrin sponge, gelatine sponge, coagulated blood, dessicated serum and plasma, with similar results.

l. The osteoperiosteal flap is re-applied and fixed with interrupted sutures.

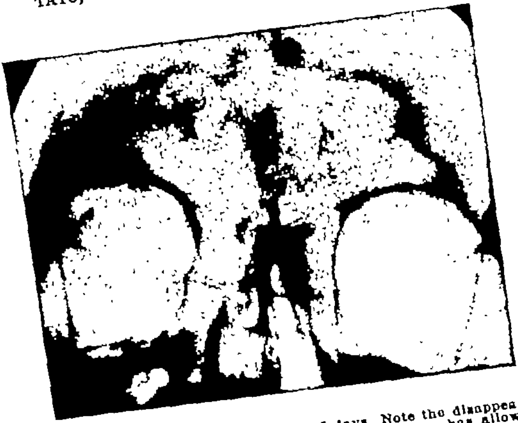

Fig. 12. Post-operative radiograph at 15 days. Note the disappearance of the septa, including intersinal septum, which resection has allowed both sinuses to be operated from one side.

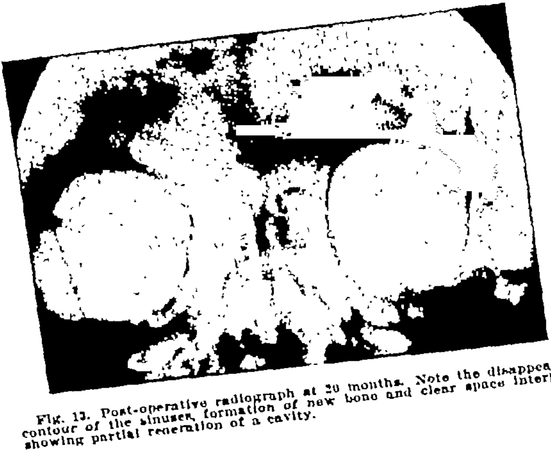

Fig. 13. Post-operative radiograph at 20 months. Note the disappearing contour of the sinuses, formation of new bone and clear space interiorly showing partial reneration of a cavity.

m. Closure of the skin and the subcutaneous tissue is done separately with interrupted sutures.

n. Application of an elastic bandage to exert a certain degree of pressure. In the cases using this technique the results show healing by first intention. This technique has been employed with good results by Professor Ermiro de Lima of Rio de Janeiro, Professor Moura of Recife, Brazil, Hidalgo of Guayaquil, Ecuador, and by Professor Bergara, Altavista, Sibbald, Romero Diaz of Buenos Aires, and others. Tieffemberg of Buenos Aires prefers inserting bone chips in the cavity and reports success in his two cases.

COMPLICATIONS.

In one of the cases, a tumefaction of moderate degree formed in the supero-internal region of the upper lid which persisted for two weeks and was found to be due to an excess of material placed inside the sinus. It disappeared progressively at the end of a year.

We have followed radiographically various cases and have proved the partial, or almost total, transformation of the bone of the cavity of the sinus, as also a repneumatization or reaeration of limited extent. As this occurred usually in the early cases, we attribute the result as probably due to an insufficient occlusion of the frontal duct (see Figs. 11, 12, and 13).

Recently Tato has operated some acute cases in the presence of complications. Beginning with an ethmoidectomy, he removed the fronto-ethmoidal wall and packed the frontal cavity with gelfoam to which sulpha powder had been added. Postoperatively he applied antibiotic therapy, and eventually the sinus filled with new bone formation. These cases are exceptional, and in no way affect the customary technique described in these pages.

ANALYSIS.

Recently it came to our notice in the "Proceedings of the Fourth International Congress of Otolaryngology" that R. R. Woods of Dublin has carried out a similar operation during the past twenty years. The technique differs in certain details from Tato's. Woods removes the floor of the sinus in all cases. Before taking away any part of the bone with a forceps he carefully separates off the mucous membrane so that the two are not included in the same bite. This is done to avoid the risk of osteomyelitis.

He removes the mucous membrane of the naso-frontal duct with a successive series of gauze strips. These are pulled down from the sinus through the nose. By gradually increasing their bulk, all the mucous membrane is finally removed, but requiring the use of some force at the end. The use of the curette is forbidden absolutely, because he believes that an infected curette scratching the endosteal surface is another potential cause of osteomyelitis.

Both Tato and Woods stress the complete and absolute removal of all the mucous membrane.

The Woods technique is made more difficult by the incomplete exposure of the high sinus, and it is a tribute to his care and patience, that, working at times blindly, and at others with the help of post-rhinal mirrors, he has been successful in removing every vestige of the mucous membrane and has never left a particle behind to form a cyst or a pocket and so defeat the object of the operation. He has had no case of osteomyelitis.

The exposure of the sinus by the osteoplastic method offers a positive advantage in the discovery of "hideouts" of mucous membrane. Tato feels that the main difference between the two techniques is the insertion of the fat, gelatine sponge, fibrin sponge, etc., to the limit of the capacity of the sinus.

A "dead space" with limited drainage is thus avoided, and if, in addition, the inserted material favors the organization of fibrous tissue by absorbing and holding blood clot, a further claim to its use can be advanced.

Postoperative chemotherapy is observed in both procedures. Woods accepts the inevitable formation of pus in the cavity after the operation, but apparently the cure of the suppuration occurs before the closure of the duct; however, Tato differs from Woods in claiming that cure by first intention is the rule governing these cases of obliteration of the sinus cavity.

SUMMARY.

1. The occlusion of the frontal sinus cavity with such materials as fat or others is described.

2. The osteoperiosteal flap technique is used in this operation, with an improvement over other similar practices.

3. The treatment of the opposite sinus after resection of the inter-sinal septum can be done without any difficulty.

4. In cases of fracture or fissure of the cribriform plate (ethmoidal fissure or fracture) with cranial hydrorrhea, we are of the opinion that this operation offers the simplest means of approach, for surgical treatment to this area, and it is easier and less dangerous than the intracranial route used by neuro-surgeons.

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THE RECIPIENT OF THE "AWARD OF MERIT" BY THE AMERICAN OTOLOGICAL SOCIETY, INC.

At its annual meeting on May 23, 1954, the American Otological Society, Inc., awarded a Silver Medal, a suitably inscribed scroll and an honorarium of one thousand dollars to Stacy R. Guild, Ph.D., of Baltimore, Maryland. The award was made in recognition of the many contributions made by Dr. Guild in the field of research in otology during the past years.

SPECIAL AWARDS.

At the 75th Annual Meeting of the American Laryngological Association held in Boston, Mass., May 27-28, 1954, Dr. Louis H. Clerf, of Philadelphia, was presented with the gold medal of the deRoaldes Award, and Dr. Henry B. Orton, of Newark, N. J., was presented with the Newcomb Award.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

A joint meeting of the North Carolina Eye, Ear, Nose, and Throat Society and the South Carolina Society of Ophthalmology and Otolaryngology will be held at Durham, N. C., November 4, 5, and 6, 1954. Headquarters will be the Washington Duke Hotel.

Guest otolaryngologists will be Dr. Stanton A. Friedberg of Chicago, Ill., Dr. John Maxwell of Ann Arbor, Michigan, Dr. George Bayling of Durham, N. C. The following guest ophthalmologists will likewise be on the program: Dr. Charles Iliff of Baltimore, Maryland, Dr. John McLean of New York, N. Y., and Dr. Townley Paton of New York, N. Y.

On Wednesday preceding the beginning of the meeting, operative clinics will be held in the various hospitals of Durham and Chapel Hill.

Finally, there will be a football game on Saturday afternoon between the University of North Carolina and the University of South Carolina in Chapel Hill.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

June 1, 1954.

Acousticon Models A-17, A-180 and A-185.

Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 1, New York.

Auditone Models 11 and 15.

Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67 and 70.

Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac Model M; Mono-Pac Model "Lyric"; Mono-Pac Model "Rhapsody."

Manufacturer: Beltone Hearing Aid Co., 2900 West 36th St., Chicago 32, Ill.

Cleartone Model 700.

Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Junior Model D-2; Dahlberg Model D-3 Tru-Sonic; Dahlberg Model D-4 Tru-Sonic.

Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Fortiphone Models 19-LR; 20A; 21-C and 22.

Manufacturer: Fortiphone Limited, Fortiphone House, 247 Regent St., London W. 1, England.

Distributor: Anton Heilman, 75 Madison Ave., New York 16, N. Y.

Gem Hearing Aid Model V-60.

Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.

Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico Model J; Maico Top Secret Model L; Maico Maxitone.

Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis, Minn.

Micronic Model 303; Micronic Model "Mercury."

Manufacturer: Audivox, Inc., *Successor to Western Electric Hearing* Aid Division, 123 Worcester St., Boston 18, Mass.

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Dahlberg Junior Model D-2; Dahlberg Model D-3 Tru-Sonic; Dahlberg Model D-4 Tru-Sonic.

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Maico Model J; Maico Top Secret Model L; Maico Maxitone.

Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis, Minn.

Micronic Model 303; Micronic Model "Mercury."

Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Model T-10; Microtone Model T-612.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 106 So. 7th St., Philadelphia 6, Pa.

Normatone Model C and Model D-53.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.

Distributor: Normatone Hearing Aid Co., 22 East 7th St., St. Paul (1), Minn.

Otarion Models B-15 and B-30; Otarion Models F-1, and F-3; Otarion Model G-3; Otarion Model H-1; Custom "5."

Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40, Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny Myte).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Model 62 Starlet; Model 72; Model 82 (Zephyr).

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Distributor: Radioear Corp., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Silvertone Model H-16, J-92; Silvertone Model P-15.

Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.

Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966; Sonotone Model 977; Sonotone Model 988.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Televox Model E.

Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 953; Telex Model 1700.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.

Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster; Model Cameo.

Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2.

Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal; Zenith "Regent."

Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices have vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TRANSISTOR HEARING AIDS ACCEPTED.

Acousticon Model A300; 1 transistor, 2 tubes. Model A-310; 1 transistor; 2 tubes. Model A-330; 3 transistors. Model A-335; 3 transistors.

Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 35, New York.

Audivox, Model 71; 3 transistors.

Manufacturer: Audivox, Inc., 123 Worcester St., Boston 18, Mass.

Beltone Concerto Model; 3 transistors.

Manufacturer: Beltone Hearing Aid Co., 2900 W. 36th St., Chicago 32, Illinois.

Maico Transist-Ear, Model O; 3 transistors.

Manufacturer: The Maico Company, Inc., 21 N. 3rd St., Minneapolis 1, Minnesota.

Micronic "All American" Hearing Aid; 3 transistors.

Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Model T1 (Red Dot); 3 transistors. Microtone Model T1 (Yellow Dot); 3 transistors. Microtone Model T31 (Micro-Mite); 1 transistor and 2 tubes.

Manufacturer: The Microtone Corporation, Ford Parkway on the Mississippi, St. Paul 1, Minn.

Otarion Model C-15; 1 transistor, 2 tubes. Otarion Model D-1; 3 transistors. Otarion Model F-22; 1 transistor, 2 tubes.

Manufacturer: Otarion, Inc., 4757 N. Ravenswood Ave., Chicago 40, Ill.

Radioear Model 820; 3 transistors.

Manufacturer: E. A. Myers & Sons, Inc., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Silvertone Model H-25; 3 transistors.

Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.
Distributor: Sears, Roebuck & Co., 925 South Homan Ave., Chicago 7, Illinois.

Sonotone Model 1010; 1 transistor, 2 tubes. Model No. 1111; 3 transistors.

Manufacturer: Sonotone Corporation, Elmsford, N. Y.

Telex Model 954; 1 transistor, 2 tubes. Telex Model 956; 3 transistors.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Unex Model TR-3D; 3 transistors.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Zenith Model Royal-T; 3 transistors. Zenith Model Super Royal-T; 3 transistors.

Manufacturer: Zenith Radio Corp., 5801 W. Dickens Ave., Chicago 39, Illinois.

SEMI PORTABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).

Manufacturer: A. M. Brooks Co., 1222 W. Washington Blvd., Los Angeles 7, Calif.

Aurex (Semi-Portable).

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.

Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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Meeting: Hollywood Hotel, Hollywood, Fla., March 17-18, 1955.

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Vice-Chairman: Dr. Willard B. Walters.
Secretary: Dr. Jack L. Levine.
Treasurer: Dr. Russell S. Page, Jr.
Meetings are held on the third Tuesday of October, November, March and May, 7:00 P.M.
Place: Army and Navy Club, Washington, D. C.

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Meeting: Fifth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
President: Dr. J. H. Font, Medical Arts Bldg., San Juan, P. R.
Time and Place: 1956, Puerto Rico.

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Chairman of Section on Otolaryngology: Max E. Pohlman, M.D.
Secretary of Section on Otolaryngology: Herschel H. Burston, M.D.
Place: Los Angeles County Medical Association Building, 1925 Wilshire Boulevard, Los Angeles 57, Calif.
Time: 6:00 P.M., first Thursday of each month from September to June inclusive—Ophthalmology Section. 6:00 P.M., fourth Monday of each month from September to June inclusive—Otolaryngology Section.

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SOCIETE CANADIENNE D'OTOLARYNGOLOGIE

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Secretary: Dr. W. Ross Wright, 361 Regent St., Fredericton, N. B.
Place: Harrison Hot Springs Spa, Harrison Hot Springs, B. C.
Time: June 13-15, 1954.

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Meeting: 3rd International Congress of Broncho-Esophagology.
Time and Place: September or October, 1954, Lisbon, Portugal.

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Meeting: Lima, Peru, 1957.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY

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General Secretary: Dr. Paul Holinger, 700 No. Michigan Ave., Chicago (11), Ill.
Meeting: U. S. A., 1957.

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NEOPLASMS OF THE MIDDLE EAR AND MASTOID. REPORT OF FIFTY-FOUR CASES.*†

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(By Invitation).
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This report is based upon a clinical analysis of 54 patients with neoplastic disease of the middle ear and mastoid observed during a 35-year period from 1917 through 1952 at the University of Michigan Hospital, and St. Joseph's Mercy Hospital at Ann Arbor, Michigan. Follow up studies in each case are complete to January, 1953. Thus, the long list of failures in the treatment of these conditions is somewhat augmented; however, a painstaking clinical analysis of a substantial group of cases of this kind must necessarily be of some good purpose if only to the essayists. An open-minded review of symptom complexes, objective findings, and therapeutic responses will reveal errors of omission and commission to be avoided in future experiences of similar nature.

It is difficult to determine the incidence of new growths of the middle ear and mastoid from study of the various series which have been reported because uniform criteria obviously have not been used. In some series, the ratio has been based on the number of otitic neoplasms compared to the total number of patients seen in a clinic for ear diseases. In others, the total number of general hospital registrations has been employed for comparison. The type of institution reporting the series may affect the ratio, since a hospital dealing entirely with malignant disease will admit a greater number of pa-

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tients with middle ear tumors than will a general hospital; also, in a referral hospital, such as the one which is the source of this report, more of these lesions will be encountered, and, therefore, a comparison of the incidence figures from the various series cannot be made unless consideration is given to the basis of their computations.

The incidence figures derived from previously reported series of cases have been quoted adequately by most writers on the subject. Some of the large series will be mentioned here, but numerous single case reports will not be included. Bezold[1] found four otic tumors in 20,000 patients with ear disease. Fraser[2] reported 15 cases of malignancy of the ear in 6,605 patients with external ear and meatal infections. Of these, only two were primary in the middle ear. Robinson[3] reported 48 new growths of the ear which were seen during a 20-year period at the Manhattan Eye, Ear and Throat Hospital, during which period a total of 212,000 patients with ear disease were seen. Of these 48 patients, four had lesions primary in the middle ear and one was a metastatic lesion. Scott,[4] in reviewing the records of the Proceedings of the Royal Society of Medicine from 1907 to 1938, found reports of 70 such tumors. Twenty-seven of these appeared to originate in the middle ear or mastoid, 15 were questionable regarding their exact site of origin, and two were definitely metastatic. Schall[5] reported 15 neoplasms of the middle ear in 90,000 patients with ear complaints examined during a 12-year period. Precechtel[6] reported seven cases of malignant tumor of the middle ear in 146,000 clinic patients. Figi and Hempstead,[7] found 48 patients with neoplasms of the middle ear and mastoid during a 20-year period at the Mayo Clinic. This gave an incidence of .003 based on total clinic registrations. Grossman et al,[8] reported six cases from the University of Illinois Hospital. Mattick and Mattick,[9] found ten cases at the Roswell Park Memorial Institute for an incidence of one in 7,000 tumor cases. Towson and Shofstall,[10] reported seven cases in a two-year period at Jefferson Hospital during which time 1,883 new patient visits were recorded.

In the light of our present knowledge it is probable that another factor has influenced the incidence statistics of many of the earlier series, particularly those dealing exclusively with malignant tumors. Undoubtedly some of the malignant middle ear lesions reported in those groups were actually glomus jugulare tumors. In some of the series, such as those of Mattick and Mattick,[9] Lattes and Waltner,[11] Winship, *et al*,[12] tumors previously described as angiosarcoma, hemandothelioma, or hemangioendothelial sarcoma have been reclassified as glomus jugulare tumors.

The neoplasms in this series have been grouped as in Table I. The inclusion of all tumors of the external canal and pinna would have increased the number of cases greatly, but since they usually present different problems, they are excluded from this report.

TABLE I.

CLASSIFICATION OF NEOPLASMS OF THE MIDDLE EAR AND MASTOID.

Group	Number of Cases
Malignant Neoplasms	
I Carcinoma (Intrinsic)*	23
II Carcinoma (Extrinsic)**	12
III Sarcoma	4
Benign Neoplasms	
IV Glomus Jugulare Tumors	10
V Neurofibroma	3
VI Fibrocystic Bone Disease	2
Total Cases	54

* Primary in the middle ear, mastoid, or inner 1/3 of external canal and inseparable from those primarily in the middle ear.

** Primary on the auricle or surrounding area with involvement of middle ear or mastoid by extension or metastatic spread.

Of the 54 cases herewith reported, 45 were treated at the University of Michigan Hospital. During the 35-year period of this study, approximately 350,000 patients were seen in the Department of Otolaryngology. There is, therefore, an incidence of one tumor of the middle ear or mastoid to each 7,777 otorhinolaryngological patients seen in the Department; however, during this period the diagnosis of "chronic suppurative otitis media" was made on 7,287 patients. Thus, an incidence of one case of carcinoma in 208 cases of chronic suppurative otitis media is obtained for this series.

Previous reports indicate that malignant neoplasms of the middle ear and mastoid conform to the general rule that carcinomata occur more commonly in older age groups while the sarcomata are more frequently seen in children. Figi and Hempstead,[7] in their series found 79 per cent of the patients with carcinoma between 40 and 70 years of age. Mattick and Mattick,[9] reported five out of eight patients with carcinoma over 40 years of age. The figures obtained in the present series would substantiate the findings of others in that 29 out of 35 patients with carcinoma were over 40 years of age (see Table II). All four patients with sarcoma were younger individuals, three of whom were less than eight years of age. The glomus jugulare tumors were confined to individuals between 40 and 45 years old, except one, a woman aged 30. The other benign neoplasms occurred for the most part in individuals less than 30 years old.

TABLE II.
AGE DISTRIBUTION.

	Group	Total Cases	Youngest	Oldest	Number Over 40 Years
I	Carcinoma (Intrinsic)	23	18	75	18
II	Carcinoma (Extrinsic)	12	34	68	11
III	Sarcoma	4	4	18	0
IV	Glomus Jugulare Tumors	10	30	54	9
V	Neurofibroma	3	23	31	0
VI	Fibrocystic Bone Disease	2	15	52	1

The sex distribution of these neoplasms has not been constant according to previous reports. Robinson,[3] reported 19 males and five females in a series of 24 cases. Figi and Hempstead,[7] found the incidence to be equally divided between the sexes in their series of 48 patients. Mattick and Mattick,[9] reported eight females and two males in their series of ten cases of neoplasm. In our series, the sex distribution is presented in Table III. In all groups, the incidence is quite equally divided between the sexes, except in group IV, the glomus jugulare tumors in which the number of females (seven) compared to males (three) gives a ratio of over two to one, which may be significant; however, the total number of cases is small, and the reliability of such a ratio must remain in doubt until other series of sufficient numbers to warrant comparison have been recorded.

From all information available, the etiology of middle ear neoplasms remains obscure. The one most constant feature of the disease has been the presence of purulent aural discharge in the majority of patients with carcinoma; however, long continued (more than two years) suppuration was present in less than one-half of the patients in this series. Many previous investigators have felt that prolonged irritation secondary to chronic suppurative otitis media was the most important etiological factor in the development of a carcinoma of the middle ear. An analysis of the cases in this series indicates strongly that other factors must be involved.

TABLE III.
SEX DISTRIBUTION.

Group	Total Cases	Female	Male
I Carcinoma (Intrinsic)	23	13	10
II Carcinoma (Extrinsic)	12	4	8
III Sarcoma	4	2	2
IV Glomus Jugulare Tumors	10	7	3
V Neurofibroma	3	2	1
VI Fibrocystic Bone Disease	2	0	2

The neoplasms described here include squamous cell carcinomas, basal cell carcinomas, adenocarcinomas, sarcomas, *glomous jugulare tumors, neurofibromas, and fibro-osteomas* (see Table I). Other types of tumors of the middle ear and mastoid have been reported by various authors. Zimmerman.[13] reported a case of Ewing's sarcoma of the mastoid, and found ten instances of this lesion reported in the literature. Peele and Hauser,[14] reported one case of Brooke's tumor of the middle ear (epithelioma adenoides cysticum). Karatay,[15] reported one case of rhabdomyosarcoma. Watanbe,[16] reported a dermoid in the mastoid antrum. Snyder,[17] reported a giant cell tumor of the mastoid.

Of the carcinomata the most common is the epithelioma or squamous cell carcinoma. Many investigators feel that such growths must originate in the depths of the external canal and proceed by direct extension to the middle ear. Undoubtedly, this is the mode of development in many cases; however, the fact that there is an occasional squamous cell carcinoma found in the middle ear or mastoid without apparent involvement of the epithelium of the external canal, makes it reasonable to assume that such a lesion may have its origin in the squamous epithelium that has invaded the tympanum after long continued suppuration or, perhaps, even after metaplastic changes have occurred in the tympanic mucosa.

The basal cell carcinomata, which are seen rather frequently involving the auricle and surrounding skin, may invade the middle ear and mastoid by direct extension. No case of a primary basal cell carcinoma of the tympanum was found in the literature reviewed or in this series.

Primary adenocarcinoma of the middle ear is exceedingly rare, One case in this series was reported by Furstenberg,[18] in 1924 as the second instance of such a tumor on record at that time. The first to be recorded was by Lange.[19] These tumors supposedly develop from the mucous glands of the tympanum. Rarely, a metastatic adenocarcinoma may be found in the tympanum or mastoid.

The sarcomata, which are decidedly more rare than the carcinomata, may develop from the mucoperiosteum of the tympanum and mastoid or even from the dura. Various types of these tumors may be encountered, but most often, they are of the spindle cell variety. Many of the tumors previously placed in this category, such as the angiosarcomas and hemangioendothelial sarcomas, have now been reclassified as glomus jugulare tumors. Sarcomas usually are seen in children although Friedenwald and Kemler,[20] reported a case of spindle cell sarcoma in a 42-year-old man.

The glomus jugulare tumors are a more recent addition to the general classification of middle ear tumors. The work of Guild,[21] in describing the glomus jugulare and the correlation of this purely anatomical structure with a clinical situation by Rosenwasser,[22] are well known. More recently, the term chemodectoma has been proposed by Mulligan,[23] as a more accurate and descriptive designation of these neoplasms. The histological picture of these tumors with the numerous capillaries and dilated blood spaces explains their great vascularity. The low incidence of mitotic figures would suggest a rather benign type of tumor with little tendency to metastasize, a fact which has been substantiated clinically. In this report, these lesions are classified as benign tumors although it would seem that there may be malignant variants since one patient with metastasis to cervical nodes was reported by Winship, *et al.*,[18] and one patient with metastasis to the liver was reported by Lattes and Waltner.[21] One patient included in this series previously had a lesion removed from the buccal mucosa which on histological examination proved to be identical with the middle ear neoplasm.

More recent investigations by Guild,[24] have shown that glomus formations may be present normally in locations other than that originally described. Guild has noted such paraganglionic tissues along Arnold's nerve as far peripherally as the descending part of the facial canal. These observations have helped to explain the occurrence of glomus jugulare tumors in unusual situations as in one case of this series in which the tumor was located beneath the mastoid tip and found to involve the facial nerve at the stylo-mastoid foramen.

Since the original description of the glomus jugulare tumors by Rosenwasser,[22] many cases of this condition have been recorded either as initial reports or after reclassification of tumors previously diagnosed as neoplasms of other types such as the publications of Kipkie,[25] Le Compte, *et al.*,[26] Winship, *et al.*,[18] Iattes and Waltner,[21] Lundgren,[27] DeLisa,[28] Dockery, *et al.*,[29] Lewis and Grant,[30] Poppen and Riemenschneider,[31] and Tamari, *et al.*[32]

Tumors of the facial nerve occurring along its course through the temporal bone have been included in our series because clinically they are essentially middle ear tumors. The three cases included in this series are neurinomas. Other reports of this lesion have been presented by Williams and Pastore,[33] Rosenwasser,[34] Tremble and Penfield,[35] Roberts,[36] who found 12 other cases in reviewing the literature, and Maxwell.[37] Guttman and Simon,[38] and Figi and Hempstead,[7] each reported a case of neurofibrosarcoma of the facial nerve. The site of involvement in the majority of the cases was in the vertical course of the facial nerve; however, in one case in this series, the tumor involved the intratympanic segment. In the report of Tremble and Penfield,[35] the tumor was located just distal to the geniculum along the course of the greater superficial petrosal nerve.

Osteomas of the mastoid have been included in this report to show the wide variation which may occur in tumors of the same cell type occurring in this location. The usual picture of an osteoma is a well circumscribed dense tumor of compact bone occurring in an otherwise normal mastoid. Such tumors have been reported by Coates,[39] Friedberg,[40] Hempstead,[41] Cinelli,[42] Stuart,[43] Simpson,[44] Jervey,[45] and Marrocco.[46] Since these localized tumors in the mastoid have been reported rather infrequently, it would seem that their occurrence here is much less common than in the paranasal sinuses. In contrast to the well circumscribed tumors just mentioned, a more widespread involvement of the temporal bone may be encountered as in one case of this series which demonstrated an extensive diffuse involvement of the temporal bone by a fibro-osteoma. Although the neoplasm was in the nature of fibrocystic bone disease or a localized Paget's disease, the histopathological structure was similar to that seen in the circumscribed osteomas. The terms osteoma, osteo-fibroma, fibro-osteoma, or ossifying fibroma all have been used in describing these tumors. Exostosis and pedunculated osteomas in the external auditory canal have not been included in this report.

The clinical diagnosis of a neoplasm of the middle ear presupposes a painstaking objective examination of the external canal and tympanic membrane as well as the functional testing of the cochlear and vestibular mechanisms in conjunction with roentgenographic studies.

Pain, out of all proportion to the apparent extent of bone disease or amount of purulent discharge, is one of the more common symptoms (see Table IV). This was noted by Figi[7] (pain in 33 of 48 patients); Mattick and Mattick[9] who reported pain in all eight of the carcinomas observed by them; Scott,[4] who maintained that pain was the most common presenting symptom; Robinson,[8]; Barnes,[47]; Ward, *et al.*,[48]; Thorell,[49]; and Fraser.[2] Bowman,[50] however, expressed his belief that pain occurs late in the disease.

Bleeding from apparent granulation tissue should make one suspicious of malignancy, particularly if the duration of the chronic suppurative otitis media as evidenced by the presence of foul smelling discharge is of short duration. Any aural

TABLE IV. SYMPTOMATOLOGY.

Group	Total Cases	No Middle Ear Suppuration	Otitis Media Two Years or Less	Chronic Otitis Media More Than Two Years	Pain	Facial Paralysis
1 Carcinoma (Intrinsic)	23	1	11	11	18	8
II Carcinoma (Extrinsic)	12	3	5	4	7	5
III Sarcoma	4	1	8*	0	2	2
IV Glomus Jugulare Tumors	10	6	2	2	3	2
V Neurofibroma	3	2	0	1	0	3
VI Fibrocystic Bone Disease	2	2	0	0	0	0

* Less than 3 months.

polyps occurring in a chronic suppurative otitis media should be removed and subjected to histopathological examination. Such advice must seem extremely elementary to the trained otologist; however, carelessness in disposing of such polyps has been responsible for delayed final diagnosis in too many instances.

Facial paralysis, labyrinthine irritation and perceptive deafness occurring in an individual with chronic suppurative otitis media are indications of extension of the disease beyond the confines of the tympanic cavity and pneumatic structure of the mastoid. When associated with a malignant lesion, it would seem that the findings are more valuable from a prognostic rather than a diagnostic viewpoint because they indicate extensive bony infiltration. Since the intratympanic segment of the facial nerve is more vulnerable than the otic capsule, a facial paralysis is a common finding and may occur rather early in primary middle ear carcinomas, as indicated by Figi,[7] Mattick and Mattick,[9] Grossman,[8] Robinson,[3] Keeler,[51] Barnes,[47] Towson and Schofstall,[1] and Schall.[5] In our series, the occurrence of facial paralysis is indicated in Table IV.

In brief, there are no characteristic early clinical manisfestations in carcinoma of the middle ear. Unreasonable delay in making a diagnosis may be avoided, however, in most instances if exuberant granulations are removed and examined microscopically. Cytological examination of the purulent exudate as advocated by House,[52] and Friedman[53] may be of value in many cases. This method of diagnosis was not used in any patient in our series.

Sarcomas of the middle ear frequently defy early diagnosis because of their extremely rapid growth. A rapidly enlarging aural polyp associated with purulent discharge which is malodorous from the onset in a child, should excite suspicion of the presence of a neoplasm. In these patients, early extension through the bone producing complications is commonly encountered.

The early diagnosis of benign tumors of the middle ear presents a real challenge to the otologist and demands painstaking study and the use of every possible diagnostic aid.

The classical glomus jugulare tumor, arising as it does from the region of the jugulare fossa, grows slowly to fill the middle ear and to invade the temporal bone. The conductive deafness, discolored tympanic membranes, and absence of diagnostic radiographic findings in these cases have been mistakenly attributed to eustachian tube closure and secretory otitis media (see Fig. 1.).

Fig. 1

An example of an extensive glomus jugulare tumor without evidence of diagnostic radiographic findings.

In such instances, palpation of the tympanic membrane with a flexible silver probe to reveal a rather rubbery firmness, quite unlike the soft fluctuating membrane which encloses fluid, has been most helpful. Although myringotomy and biopsy in such instances are hazardous because of hemorrhage, they are justified in selected doubtful cases. A diagnosis was established in this manner in three of the patients

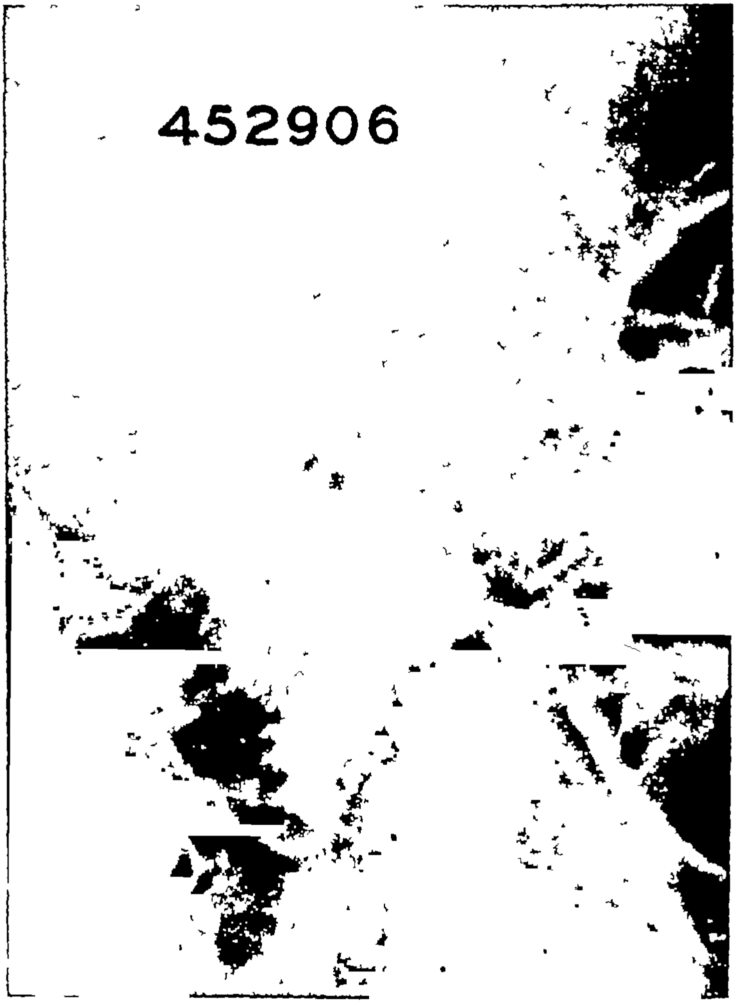

Fig 2

An example of an extensive glomus jugulare tumor showing radiographic evidence of bone destruction in our series after inspection and palpation of the tympanic membrane led to the clinical diagnosis of middle ear tumor. In no instance was hemorrhage uncontrollable although it

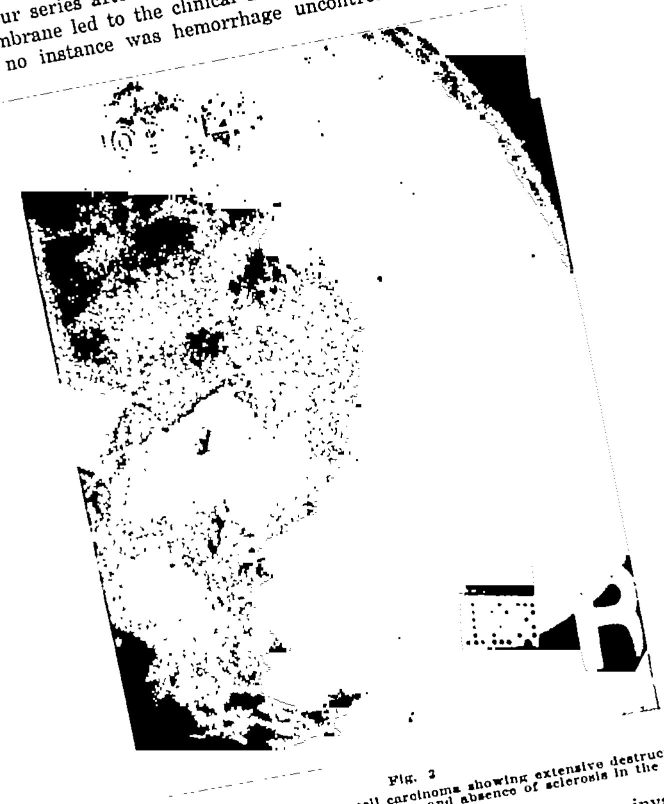

Fig. 2

An example of a squamous cell carcinoma showing extensive destruction with evidence of irregular infiltration and absence of sclerosis in the surrounding bone

was severe in each patient. Not until the tumor has invaded the bone extensively will the radiographs be of diagnostic value (see Fig. 2). By this time, the neoplasm in the middle ear is usually quite apparent.

Occasionally, a small tumor may be visualized through an intact and transparent tympanic membrane. The diagnosis of a solitary neuro-fibroma of the intratympanic segment of the facial nerve was suspected by such an observation in one

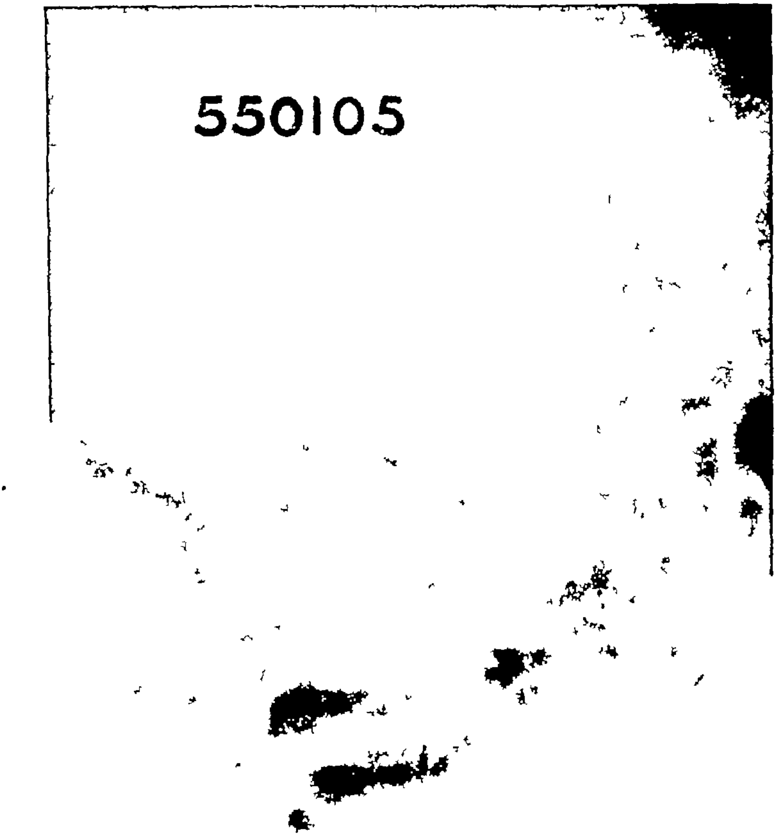

Fig 4

An Hand-Schuller-Christian's disease showing areas of de-
structio characteristic smooth clearcut border

'en ies. The clinical diagnosis was substan-
d Cranmer of Toledo, Ohio, who resected
rted a nerve graft.

Osteomas, unless large enough to present external tumefaction, are usually discovered accidentally in roentgenograms of the skull or mastoid. In this region, it is exceedingly rare for them to become large enough to produce symptoms due to pressure on vital structures.

A discussion of the differential diagnosis of neoplasms of the middle ear and mastoid should include consideration of Hand-Schüller-Christian's disease which occasionally involves the temporal bone. This condition occurs here much more frequently according to our experience than does the eosinophilic granuloma or the other variants of lipid histiocytosis. The radiographically demonstrable area of destruction simulates a malignant lesion chiefly because of the absence of sclerosis in the surrounding bone (see Fig. 3); however, the border is smooth and clear cut without evidence of irregular infiltration which characterizes the malignant tumors (see Fig. 4). Since these lesions often occur also in other locations, the diagnosis may be made after full skull X-rays are taken as well as films of the spine, pelvis and long bones (see Fig. 5).

On occasions, tuberculosis of the middle ear may run a clinical course with early manifestations simulating those of a malignant neoplasm as stated by Bowman.[50]

No form of treatment of these malignant neoplasms has been highly successful. When the hemangioendothelioma, the angiosarcomata and other tumors which most probably were glomus jugulare tumors are excluded, the results of therapy have been generally poor.

Radical surgical excision and irradiation therapy have been the only therapeutic procedures of any value. The combination of these two forms of therapy has been advocated by the majority of previous investigators; for example, Schall,[8] Thorell,[48] Figi and Hempstead,[7] and Mattick and Mattick.[9] Figi and Hempstead[7] favored electro-coagulation and electrocautery rather than sharp dissection excision. They felt also that direct implantation of radium into the cavity should be used in addition to external radiation. Ward, *et al.*,[44] have advocated radical mastoidectomy with neck gland dissection in continuity for each patient. In addition, if infiltration of the external canal is extensive, or if the pinna is involved,

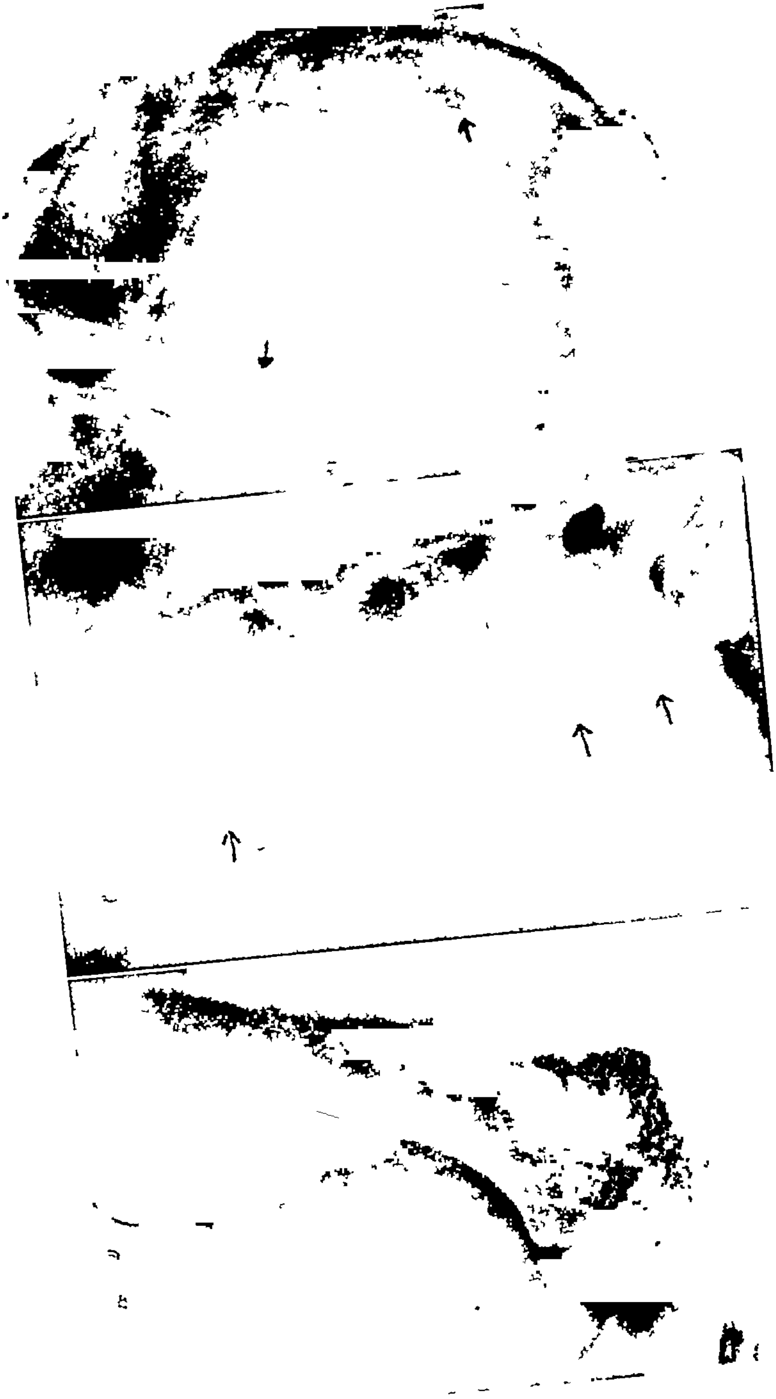

Fig 5

Examples of Hand-Schuller-Christian's disease showing the charac
ation in the skull, spine, and humerus

they recommend that the parotid gland be removed with sacrifice of the facial nerve. They suggest further that the condyloid process and ramus of the mandible may have to be included. These authors presented a series of five patients with squamous cell carcinoma, all of whom were treated by a radical resection of the neoplasm plus radical neck gland dissection. Lymph nodes showing carcinoma were found in all the specimens although these nodes were not demonstrable on clinical examination pre-operatively.

An analysis of the results of therapy in our series is presented in Table V. The treatment utilized was radical surgical excision of the neoplasm whenever possible. In the majority of patients intensive roentgen therapy was administered post-operatively. In most instances, external irradiation was used, but in a few patients, radium was implanted into the cavity. In nine patients with very extensive highly anaplastic neoplasms, irradiation therapy alone without surgical excision was considered to be the treatment of choice.

In contemplating the radical approach advocated by Ward, *et al.*,[44] one must consider not only the possibility of metastasis to cervical nodes but also the possibility of extension through the peri-tubal lymphatics, the invasion of the petrous apex, and the involvement of the dura. In those individuals with palpable metastatic cervical nodes secondary to neoplasms primary in the tympanum, deep extension by way of peritubal lymphatics is most likely. Since removal of the involved cervical nodes would be ineffectual in such a condition. the advisability of the routine radical block resection is doubtful. The primary site of the lesion and the extent of involvement of the middle ear and temporal bone must determine the type of operation to be employed in each instance.

To date, the use of Roentgen therapy alone has produced disappointing results. Newer techniques for delivering maximal concentration of irradiation to the involved region give promise of further help from this form of therapy.

If any one factor is most responsible for the ineffectiveness of therapy, it would seem to be the delay in establishing a diagnosis. The majority of patients reported here were not

TABLE V. TYPE OF TUMOR AND PRESENT STATUS.

Group	All Types		Present Status
I Carcinoma Intrinsic)	Squamous cell carcinoma Adenocarcinoma	21 2	18 — dead from neoplasm 2 — living and well with no recurrence 5 years and 32 years post-op. 2 — operated during past year and are being followed in clinic. 1 — died of another cause 11 years post-op.
II Carcinoma (Extrinsic)	Squamous cell carcinoma Basal cell carcinoma Squamous and basal cell Scirrhous carcinoma	8 2 1 1	12 — dead from neoplasm
III Sarcoma	Spindle cell fibrosarcoma *Angiosarcoma Lymphosarcoma Highly malignant sarcoma	1 1 1 1	4 — dead from neoplasm
IV Glomus Jugulare		10	8 — living and well 4 months to 14 years after treatment 2 — dead from unrelated causes
V Neurofibroma		3	3 — living and well with no recurrence
VI Fibrocystic Bone Disease		2	2 — living and well with no recurrence

* Female, age 7, with pulmonary metastases.

diagnosed early. Many had been treated by local forms of conservative therapy for months before a suspicion of malignancy was aroused.

The treatment of the benign tumors, as might be expected, has been highly successful. All but two of the glomous jugulare tumors in this series have been removed surgically. Roentgen therapy was used as an adjuvant measure in the earlier cases which had been diagnosed malignant lesions. Only two patients were treated with X-ray therapy alone: one, an elderly woman whose general condition contraindicated a major operation, and the other a younger woman who refused operation. Although clinical and roentgenographic evidence indicate little response to this treatment, both of these patients are living, one, 15 years after treatment. Whether the natural slow growth of the tumor has been retarded is not known.

Surgical removal of a glomus jugulare tumor should be undertaken with an appreciation of the hemorrhage likely to be encountered during the procedure. Extensive removal of the lesion with adequate blood replacement during the operation will effect a cure in the majority of patients. Intensive irradiation after operation may be of some value.

The neurofibromas of the facial nerve should be removed surgically, and the nerve repaired by the use of free graft whenever possible. A graft 63 mm. long, having five branches, was utilized in one patient in this series whose nerve was involved in tumor from the geniculate ganglion to the branches in the parotid gland. A satisfactory functional result was obtained. The great auricular nerve from the cervical plexus is a convenient nerve to use for this purpose.

The osteomata, both the circumscribed and the diffuse fibrocystic types, are surgical problems. The circumscribed lesions seldom present symptoms requiring treatment; however, when indicated, excision usually can be accomplished without difficulty. Removal of the diffuse lesion may present many technical difficulties due to extensive invasion of the temporal bone.

SUMMARY.

Fifty-four patients with neoplasms involving the middle ear and mastoid treated at the University of Michigan Hospital and St. Joseph's Mercy Hospital in Ann Arbor, Mich., during the 35-year period from 1917 to 1953 have been subjected to clinical analysis.

In the group of carcinomas, no characteristic symptom complex was elicited which might be of value in making an early diagnosis. Although pain was the presenting symptom in 18 patients, there was little indication that it was an early manifestation of the disease. When associated with foul smelling discharge of insidious onset and short duration (one year or less), it seems to have definite significance.

Eight patients in the "intrinsic carcinoma" group had facial paralysis. In six instances, the onset was closely correlated with the beginning of pain.

No significant data have been collected to permit conclusions regarding the etiological relationship between chronic supppurative otitis media and carcinoma of the middle ear; however, it would seem doubtful, from this study, that chronic suppuration in the middle ear is a highly important factor for the following reasons:

1. The incidence of carcinoma in individuals with chronic suppurative otitis media is low. The incidence in this study of one case of carcinoma in 208 cases of chronic suppurative otitis media certainly does not indicate an acceptable ratio because patients are admitted to University Hospital on a referral basis only. This means that the patient has presented some unusual diagnostic or therapeutic problem to the referring physician. A higher incidence of uncomplicated chronic suppurative otitis media might be expected in a hospital other than the referral type.

2. There was a definite history of chronic middle ear disease of more than two years' duration in only eleven (less than one-half) patients.

3. Carcinoma in association with cholesteatoma is exceedingly rare. One would scarcely expect this to be true if chronic irritation of the lining epithelium of the middle ear and mastoid were conducive to the development of a carcinoma.

Despite radical surgical excision used in association with intensive irradiation therapy, the prognosis in carcinoma of the middle ear and mastoid is very poor.

The twelve patients in the "extrinsic carcinoma" group all died from their neoplasms. The "intrinsic carcinoma" group, which numbered 23 patients fared but slightly better. Eighteen died of their neoplasms in one month to three years. Two are living and well five years and 32 years post-operatively. One died of unrelated causes 11 years post-operatively, and two are alive but less than two years after operation. If the prognosis is to be improved, earlier diagnosis is mandatory. This can only be accomplished by more careful observation and intelligent management of patients with discharging ears. Granulations must be subjected to microscopic examination and carcinoma suspected when bleeding tissues are associated with foul smelling purulent discharge of insidious onset and short duration. Cytological examination of aural discharge deserves further study.

Sarcomas of the middle ear are rare, tumors occurring more commonly in children. They offer a poor prognosis. Three of the four individuals in this series so afflicted were less than eight years old and one was 18 years of age. The sarcomas were of various types. All patients died within two years after the diagnosis was made.

Of the benign tumors, the glomus jugulare type is the most common. Ten instances of this neoplasm are recorded in this report. Three of these have been diagnosed and treated since this tumor was described as a clinical entity. Seven were operated upon prior to this time and were originally diagnosed malignant neoplasms.

The prognosis of patients with glomus jugulare tumors is generally good after radical mastoidectomy, although severe hemorrhage may render the operation most difficult. Of the ten cases in this series, eight are living and well and under periodic observation. Two have died of unrelated causes.

The three neurofibromas in this series were apparently of the solitary type and not associated with Von Recklinghausen's disease. Two had facial paralysis. In two instances the tumor was well enough localized to permit excision and facial nerve repair. As previously reported by one of us, neurofibromatosis of the facial nerve distal to the Fallopian canal is not likely to produce facial paralysis.[37]

Two instances of fibrocystic bone disease are recorded: one was an extensive diffuse lesion diagnosed as an osteofibroma; the other was a circumscribed dense tumor clinically simulating a true osteoma, but the pathological report was that of a fibro-osteoma.

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THE STUDY OF EPITHELIAL CILIA AND SPERM FLAGELLA WITH THE ELECTRON MICROSCOPE.[*][†][‡][§]

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The importance of cilia in the physiology of the nose, paranasal sinuses and lower respiratory tract seems more widely appreciated by clinical otorhinolaryngologists than it is by professional physiologists who as a rule devote very little text-book space to a consideration of ciliary motion. Many biologists have been actively interested in cilia not only because of their widespread occurrence and varied functions in the animal kingdom, but because of a belief that an analysis of the mechanism of ciliary motion held the key to an understanding of all contractile protoplasm. Perhaps it does, indeed, but the assumption that those "optically homogeneous motile cell processes" represent a very *simple* form of contractile protoplasm is not borne out by recent observations with the electron microscope.[1,2,3] On the contrary, each cilium is found to have a highly complex and precisely ordered internal structure that has escaped detection with the light microscope.

As seen with the conventional light microscope, the components of the ciliary apparatus of epithelial cells are the cilia, their basal bodies, situated just within the distal border of the cell, and, in certain instances, fine fibrous rootlets which seem to arise from the basal bodies and pass downward for

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[†] Many of the observations reported here were made while the author was a Visiting Investigator at the Rockefeller Institute for Medical Research, in the Laboratory of Dr. K. R. Porter. A detailed account of this phase of work has been published in the Journal of Morphology, Vol. 94, pp. 221-282, 1954.

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[§] John and Mary R. Markle Scholar in Medical Science.

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some distance into the apical cytoplasm. As a rule, the cilium itself appears optically homogeneous, but by using special staining methods a few of the early cytologists described a single axial filament or core running lengthwise of the cilium.[4] Later workers, taking advantage of polarizing and dark-field microscopy, suggested the presence of several delicate fibrils instead of a single one,[5,6] but this concept of the fine structure of cilia was not accorded general acceptance until the late 1940's when multiple filaments were demonstrated by means of the electron microscope in protozoan cilia and metazoan sperm flagella.[7] These early electron microscope studies used intact or partially fragmented cellular material simply dried onto the specimen grid. Consequently, it was impossible to determine with accuracy the number or the arrangement of the longitudinal filaments and no observations could be made upon the intracellular components of the ciliary apparatus. In the past five years satisfactory methods have been developed for embedding tissues in plastic and for cutting sections less than a twentieth of a micron in thickness.[8,9] These advances now make it possible to preserve the normal spatial relations of tissue components and to study the fine architecture of cells down to structures only a few macromolecules in thickness. The present paper presents some results of the application of these methods to a comparative study of ciliated epithelia derived from a wide sampling of the animal kingdom including an annelid worm, two varieties of molusks, two species of amphibia and three different mammals including man.

OBSERVATIONS.

The intestinal epithelium of the fresh water mollusk *Elliptio* is particularly favorable material in which to study the intracellular portion of the ciliary apparatus for in this and other invertebrate forms the rootlets of the cilia reach their greatest development. Arising from rows of refractile basal bodies just beneath the striated cuticular border, the rootlets, converge to form a conspicuous cone of fibers with the apex at the level of the nucleus. Opinion has varied as to their function. Some investigators have thought that they were merely anchoring structures serving to stabilize the basal bodies against the vigorous lashing of the cilia. Others have considered them contractile elements concerned with ciliary movement, and still others have regarded the rootlets as structures of neural or trophic function. When these cells are examined with the electron microscope, several new details of fine structure are observed even at relatively low magnifications. The cuticular border is comprised of a very great number of slender, vertically oriented cell processes (80 mμ wide and 1.5 μ long) in closely-packed parallel array (see Fig. 1A). These processes are doubtless responsible for the refractility of this border and the appearance of vertical striation noted with the light miscroscope. The cilia pass through this thick pile or nap of shorter processes constituting the cuticular border

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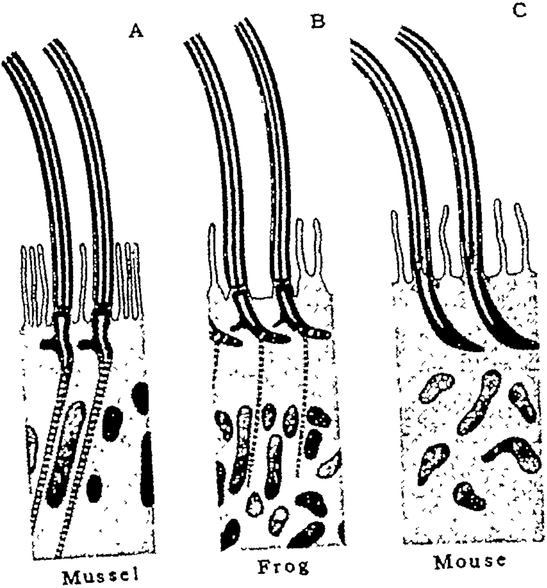
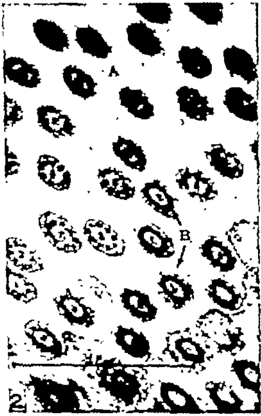


Fig. 1. Diagrammatic comparison of the structure of the ciliary apparatus in an invertebrate, an amphibian, and a mammal. Note the asymmetrical shape of the basal bodies when cut in the plane of ciliary beat, and the progressive reduction in size of the cross-striated rootlets, from invertebrate to mammals. In mammals the longitudinal filaments of the cilium are continuous with the substance of the basal body.

and project several microns above its surface. The shaft of the cilium contains a bundle of longitudinal fibrils which are embedded in a homogeneous protoplasmic matrix, and this in turn is surrounded by a ciliary membrane which is continuous with the cell membrane. The longitudinal filaments are straight and parallel and have no detectable periodic cross-striation along their length. They do not pierce the basal body and continue into the cytoplasm as has been suggested heretofore, but they end abruptly in a dense transverse plate fixed to the flattened upper surface of the basal body. The basal body is asymmetrical and has a dense cortex and a core of relatively low density which gives it the appearance of being hollow. A fiber connecting successive basal bodies in the same row has not been observed, but a blunt process does project from each basal body in a horizontal direction. The alignment of these processes is probably responsible for the appearance with the light microscope of a continuous line joining the basal bodies. Two rootlet fibers arise from the lower pole of each basal body and diverge at an angle of 10 to 15 degrees in the plane at right angles to the direction of beat. (Thus the two fibers are not seen in the lateral view presented in Fig. 1A). The rootlet fibers show a regular cross-striation with a major repeating period of about 65-70 mμ along their length. Although the major period is similar to that of collagen, the spacing of the finer intraperiod structure seems to distinguish this fiber from collagen and other extracellular fibrous proteins which have been studied to date.

When the cilia are examined in cross section, the longitudinal filaments in their interior are found to have a constant arrangement. One pair of filaments is situated in the axis of the cilium and nine pairs are uniformly spaced around them (see Figs. 2 and 3). The members of the peripheral pairs are so closely joined that each pair commonly appears as a single dot in the cross section, but their double nature is apparent in micrographs of good resolution taken at very high magnification. The two central filaments are less closely associated, and the orientation of these two is the same for all of the cilia on a cell. The direction of pendular motion of a cilium appears to be perpendicular to the plane passing through its two central filaments.

The structure of the ciliary apparatus in the epithelium of the pharynx or oviduct of the frog is very similar to that found in the mollusks (see Fig. 1B). Such minor differences as do exist are confined to the intracellular components. The hollow appearing basal bodies are falciform in shape and give rise to a single cross-striated rootlet. In the ciliated intermediate segment of the nephron definite vertically oriented rootlets are lacking but conspicuous, cross-striated fibers having the same periodicity are randomly oriented in the ecto-



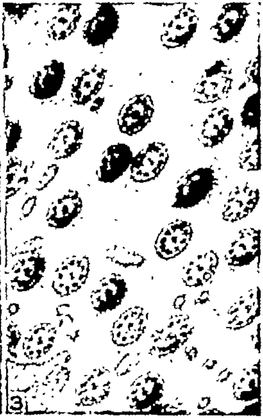


Fig. 2. Transverse sections of frog cilia showing two filaments in the center and nine pairs around the periphery (at A). Near the junction of the cilium with its basal body (at B) the outer filaments are fused together into a cylindrical structure which appears in cross section as a dense ring. Magnification 62,000 X.

Fig. 3. Sections of cilia on the epithelium of the mouse oviduct revealing the same number and arrangement of filaments as in the frog (see Fig. 2.) The identical cross-sectional appearance is found in cilia from the human Fallopian tube. Magnification 40,000 X.

plasm surrounding the basal bodies (see Fig. 5). Some of these seem to attach to basal bodies but the majority do not. It is not known whether such fibers in the ectoplasm give it contractile properties or whether their presence is merely an indication that the protoplasm there is very firmly gellated to provide a firmer supporting matrix for the basal bodies.

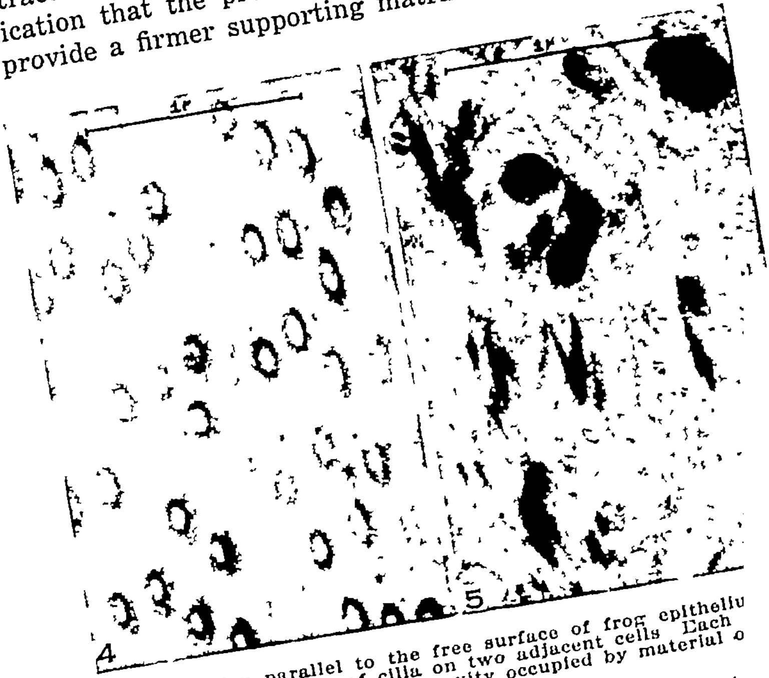


Fig 4 Section parallel to the free surface of frog epitheliu through the basal bodies of cilia on two adjacent cells Each has a dense cortex and a central cavity occupied by material o tron density Magnification 44,000 X.

Fig 5 An area of the apical cytoplasm of a ciliated cell li mediate segment of the frog nephron. Numerous cross-striat varying thickness are randomly oriented in the ectoplasm. have the same periodic structure as the vertically oriented ro cilia of the pharynx Magnification 44,000 X.

The internal organization of the free portion of in the amphibia is identical to that of the fresh lusks, having two longitudinal filaments in the nine pairs around the periphery (see Fig. 2). tangential to the cuticular border of the epith impressed by the uniformity in diameter of the large number of them on each cell. Estimate number available in the literature vary widely thor, the organ and the species studied. The dir range from 0.1 to 0.3 μ in diameter and 3.5 to 1

In a section of human nasal epithelium Lucas reported an average of 8.5 cilia on a cell surface 6.2 μ in diameter. Alcock[11] found 15 to 20 cilia per cell in nasal epithelium of the fetal pig, and Valentin, quoted by Kolliker, placed the number at 10 to 22 per cell in tracheal epithelium. Electron micrographs do not afford an opportunity to measure length because no cilium lies within the plane of a 50 mμ section for its entire length. A fairly high degree of accuracy, however, can be expected from measurements of diameter and from the enumeration of cilia per unit area of cell surface. In the epithelium of the frog oviduct the cilia were 150-175 mμ in diameter. They were arranged in rows and the number per square micron of surface averaged eight. This would mean that a columnar epithelial cell only 5 μ in diameter would carry 150 to 200 cilia. In the other animal species studied, the diameter of the cilia was approximately 200 mμ, which is somewhat larger than in the frog. One may assume the number per unit area would be correspondingly smaller, but in any case, the number of cilia per cell would seem to be greatly in excess of all previous estimates.

The majority of the experimental work on cilia has been one on epithelia from mollusks and amphibians, and physiologists and clinicians alike have cautioned against a too general application of the information derived from study of these lower forms to problems of human physiology. It is reassuring, therefore, to find that the cilia of man and other mammals depart very little from the structural organization already described for the mussel and the frog (see Fig. 1C). Two minor differences are worthy of mention. In the submammalian forms studied, the filaments in the shaft of the cilium were found to end in a transverse basal plate which was distinct from the basal body. In mouse and man, on the other hand, there is no apparent discontinuity between the cilium and its basal body; instead, the longitudinal filaments are continuous with the electron-dense material comprising the cortex of the basal body. The ciliary rootlets which were large in the mollusca and somewhat less well developed in amphibia are vestigial or entirely lacking from the mammalian epithelia which have been studied to date. In a few instances extremly delicate fibers were detectable in the apical cytoplasm, but these only occasionally were attached to basal bodies. From the progressive reduction in the degree of development of ciliary rootlets as one goes higher in the evolutionary scale, it is inferred that these intracellular fibers are not concerned with ciliary beating *per se* but they may possibly play a role in the metachronic rhythm or propagated wave motion which sweeps across the epithelial surface in lower forms but which is not commonly seen in mammalian ciliated epithelia.

In an investigation now in progress on human spermatogenesis we have had an opportunity to compare the organization of the flagella of spermatozoa with the structure of cilia. The number and arrangement of the longitudinal filaments is the same, with two in the center and nine pairs around these. A cross section of the end-piece of the sperm flagellum

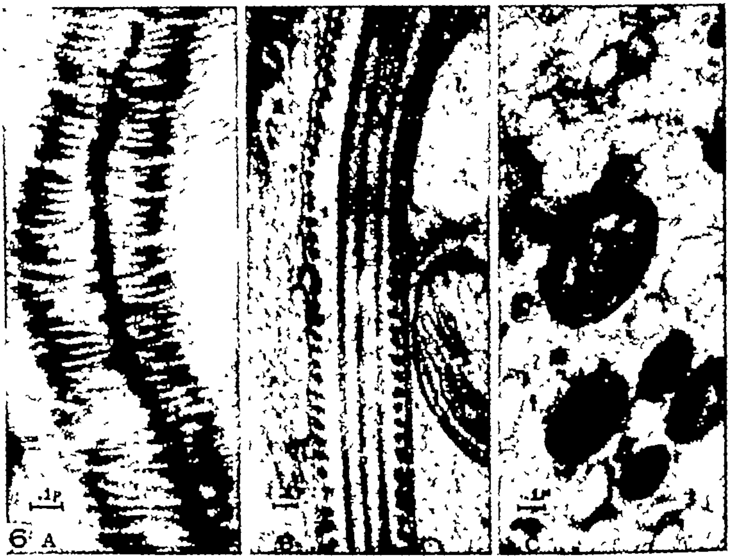


Fig. 6. (A) Section passing tangential to the surface of the principal segment of the flagellum of a human spermatozoan. The fibers of the circumferential wrapping are seen joining one of the two longitudinal densities which run along opposite sides of the flagellum. Magnification 65,000 X.

(B) A longitudinal section through the middle of a sperm flagellum showing three of the internal longitudinal filaments and on either side the cut ends of the encircling filaments. Magnification 50,000 X.

(C) A transverse section of a human sperm flagellum in the principal segment. A filamentous wrapping is interposed between the bundle of longitudinal filaments and the ciliary membrane. This component is lacking in the terminal segment of the flagellum which has a cross section just like that of a cilium. Magnification 54,000 X.

is indistinguishable from a cross section of a cilium, but throughout the principal piece a delicate filamentous wrapping is interposed between the longitudinal filaments and the enclosing membrane. A spiral winding was described and illustrated by Retzius, 1906[12] using the methods of classical cytology, and the helical nature of this structure has been re-

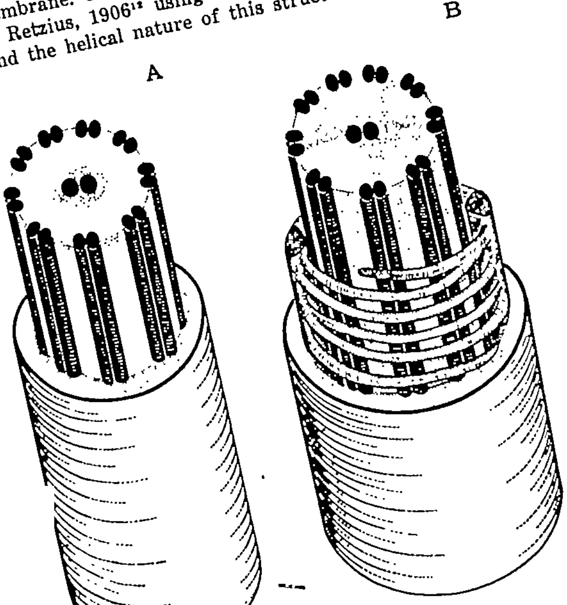


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Fig. 7. A diagrammatic presentation of the structure of a cilium (A) and the principal piece of a sperm flagellum (B). The circumferential wrapping commonly described as the "cortical helix" does not appear to be a simple, regular spiral. Occasionally the wrapping filament is seen to branch and successive turns are held together by their common attachment in two longitudinal densities situated on opposite sides of the flagellum. These components are lacking in the terminal piece of the sperm tail which has the same structure as a cilium.

affirmed on the basis of electron microscope studies on dried and partially fragmented spermatozoa.[13,14] When seen in sections tangential to the surface of the flagellum (see Figs. 6A, 6B), this wrapping seems to consist of a great number of delicate circumferential filaments held together by their common attachment to two longitudinal bands situated opposite one another in the plane passing through the two axial filaments (see Figs. 6C, 7B). In surface view the circumferential strands are seen to branch and rejoin; hence, it is doubtful whether this filamentous wrapping should be regarded as a regular helix.

DISCUSSION.

A remarkable uniformity of internal structure has been shown to exist in cilia and flagella throughout the animal kingdom with two longitudinal filaments situated in the center and nine pairs evenly spaced around them. Interestingly enough, Irene Manton[15] has shown that the same number and disposition of filaments also applies to cilia and flagella throughout the plant kingdom. Functional advantages of this particular arrangement which would account for its widespread occurrence are not immediately apparent. Lacking a satisfactory explanation one can only repeat the remark of the 18th Century comparative anatomist, Vicq d'Azyr: "Nature seems to operate always according to an original plan from which she departs with regret and whose traces we come across everywhere."

We are left in the midst of many unanswered questions. Where is the ciliary beat initiated? How is it coordinated? What role is played by the rootlets? How do cilia and flagella develop. Do they arise at the surface or are they first formed in the interior of the cell, etc.? It is hoped that the study of spermatogenesis and regenerating ciliated epithelium may cast light upon the mechanism of formation of cilia and flagella. Although observations to date with the electron microscope leave many questions unanswered they do permit us to reject a number of time-honored theories of ciliary motion, and they provide a firm morphological basis upon which new theories of ciliary motion can be erected.

SUMMARY.

The fine structure of the ciliary apparatus of epithelia in mollusks, amphibia and mammals has been reviewed, and the internal organization of the cilium and sperm flagellum have been compared.

A constant number of longitudinal filaments was found in all cilia and flagella studied. Two filaments were in the center and nine closely joined pairs evenly spaced around them. The orientation of the asymmetrical basal body and of the central pair of filaments appeared to bear a constant relationship to the direction of beat.

The ciliary rootlet fibers showed a distinct cross-banded structure with a regular repeated period of 65 to 70 mμ. The internal structure of the principal segment of the sperm tail resembles that of a cilium but has an additional component in the form of a circumferential filamentous wrapping enclosing the longitudinal filaments. Contrary to previous descriptions, this is not believed to be a regular helical winding.

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INDUSTRIAL NOISE AND OCCUPATIONAL DEAFNESS.*†

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(By Invitation).
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It is particularly fitting that a compensation administrator should discuss with a group of otologists the subjects of industrial noise and occupational deafness. Otologists have been largely instrumental in bringing these subjects to light and are to be commended for calling attention to a situation which deserves study and action. Much credit is due to the doctors for assiduously working to prevent loss of a precious human faculty, and in assisting lawmakers and administrators by evolving standards and principles upon which judgment may be passed as to measurement of and payment for hearing loss.

The otologist's interest in the subject is and should be factual, dispassionate, truly scientific. When the otologist attempts to measure and appraise loss of hearing, guided by his individual opinion as to what the law should be, and by weighing legal and economic consequences, he may although subconsciously, abandon a truly scientific role and become to a degree a partisan protagonist.

What monetary value can we place on the faculty of hearing? Prospectively, few persons would sell their hearing for any price. Helen Keller is stated to have said that if she could choose to have either hearing or sight she would prefer hearing. Retrospectively, the value of hearing is measured by many standards; one who has lost his hearing will have one standard; one who is asked to pay for loss of hearing will have another. Some will value hearing from a purely economic standpoint; others will consider social and esthetic aspects as well.

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† From the Workmen's Compensation Division, Industrial Commission of Wisconsin.

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When loss of hearing results, questions may arise as to causation, legal liability on the ground of fault or otherwise, legal value of faculty lost, and many other elements. At common law negligence must be shown on the part of someone if damages are to be collected. Not only must the plaintiff show negligence, but he must show that his loss is *caused* by an act of negligence, and that he has sustained injury in the way of pain and suffering, mental or physical harm, or social or economic loss. A court and jury will then fix the amount to be paid, not on a uniform or exact predetermined basis but by the application of multifarious and technical legal rules and principles, varying in kind from one jurisdiction to another, and susceptible to judgments and estimates of individuals, often without previous experience in deciding cases and fixing damages.

Compensation acts were designed to remove some elements of uncertainty and to give to injured employes, under specified conditions, benefits on a uniform basis, promptly, surely, and in most cases without litigation or friction. Negligence of either employer or employe is disregarded. As between states many devices have been evolved in an attempt to accomplish the principal objectives of workmen's compensation. Constantly the process continues, as by trial and error, by group pressure and persuasion, and accumulation of experience, laws and methods are created, changed, or discarded.

Should an employe be awarded something for his loss? How much can employers and consumers of products afford to pay? Is the cost of compensation in some types of cases so high that the loss to the individual is of less consequence to him and to society than is the cost to industries and consumers? What will be the result where competition exists between employers in different States with different laws?

These are questions which, under our democratic system, must be determined by each State in the light of the many interests involved and the total effect on citizens and taxpayers. Laws rarely represent absolute logic, but are evolved by compromises between demands of opposing groups.

Remarkably, the subject of loss of hearing as related to industrial noise has only rather recently assumed disconcerting proportions. We have known for years that protracted exposure to noise could and did cause loss of hearing. We have had compensation acts which, at least in terms, included such loss within its provisions; yet it is only within the last few years that claims have been made in such number as to alert and alarm industries and compensation administrators.

Scientific urge and growing emphasis on preventive medicine prompted otologists to take a greater interest in the subject and to hang up storm warnings. Labor, probably because no wage loss was actually incurred in most cases, was slow to recognize its advantage. Loss of hearing, due to continual noise, comes gradually, and is not so dramatically compelling as are accidental injuries. Industry, as in the case of silicosis, has been slow to come to grips with a problem which had not been well explored or understood.

Although laws provided for loss of hearing, claims were few and usually on the basis of accident. Question arose as to whether laws ostensibly covering gradual loss actually were effective or even constitutional.

Legislatures, not being aware of the approaching storm, did not take the initiative, nor were they pressed to make or change laws to meet the problem. It was left to unions and lawyers for claimants to discover that many potential claims were involved, calling for possible payment of large sums to many workers. Rather abruptly, many claims were filed and industry, insurance carriers, and compensation administrators awoke to the fact that here was a subject which needed prompt, earnest and continuous attention.

Within constitutional limits legislatures have the right and power to make laws which to many may seem unwise. Courts and administrators have the duty of administering laws in order to express legislative intent. They have no choice but to follow law and previous court holdings, and with the exercise of their judgment and conscience, reach their decisions. That the result may be bad from some standpoints must not deter administrators from their duty, whether that means allowance or denial of compensation benefits.

We have heard various estimates that occupational loss of hearing could result in from one billion to as much as six or seven billions of dollars of claims in this country, and that some employers and insurance carriers might become insolvent if these claims were allowed.

How valid some of these estimates may be is questionable. They have grown in the telling. Just how serious the problem really is no one of course knows. It will depend upon legislation and interpretations of laws by compensation administrators and courts. New methods of determining and measuring loss will be developed.

Much of the controversy regarding liability for certain disabilities arises because of deviation from original concepts. Whether this development is logical or desirable depends upon divergent points of view.

There probably is fairly full agreement that back in 1911 when compensation acts were first enacted in this country a primary tenet was that the benefits were to be based on wage loss. Employes themselves were to bear a substantial portion of the burden of injury. The new system was a compromise between high awards in a few cases for damages at common law and failure of many employes to collect anything at all. Benefits were not to be so large as to wreck industry or unduly burden the consumer of products. Society had a vital interest in the new legislation because if the victims of industrial injury were not compensated by other means, public or private charity would frequently have to be invoked.

An absolutely ideal compensation system would, with consummate infallibility, measure wage loss as and when it results, and at intervals pay to the person the amount provided by law.

How does it come that a different system is utilized in connection with payment for permanent disabilities? Why, if a man loses an eye or an arm, do we not follow him within time limits of law and pay him compensation as and when he can establish wage loss due to injury?

Those who are acquainted with workmen's compensation administration recognize the almost insuperable task that a system of that sort would involve.

This accounts for existence of schedules of fixed benefits in most laws. They were adopted with the commendable purpose of establishing certainty of remedy within definite limitations and as workable schemes for administration of approximate justice in myriads of cases. They are not ideal from the standpoint of accuracy and meticulous relativity. As in the case of all arbitrary classifications, they at times produce uncontemplated results.

Why does loss of hearing present such a vexatious problem in compensation administration? The answer is simple: noise is almost ubiquitous. There are so many potential claimants. Many industries produce noise which causes loss of hearing. Millions of employes are vulnerable. Few of them actually lose wages because of their partial deafness. Few, if any, of them ever become totally deaf because of prolonged exposure to noise. Ordinarily there is little reason, from the employer's standpoint, for discharge of employes from employment. Few employes wish to quit jobs which are remunerative and satisfactory to them.

Many factors present difficulty in determining the facts as to hazard and cause and extent of loss. Otologists well know that the *cause* of loss is not always easily determinable. What level and quality of noise may cause loss of hearing?

We may inquire why compensation should be paid for disabilities which may cause little if any wage loss, when funds are so urgently needed to add to the benefits paid to those who suffer substantial wage loss. Why should there be payment for 5 per cent loss of vision or for 5 per cent loss of use of an arm when only in rare cases does that disability result in wage loss?

Can industrial noise ever be reduced or eliminated so that all loss of hearing can be avoided? That something can be done will readily be admitted by the informed. Will the final attainable result be worth the price involved? We do not stop driving automobiles, although doing so might save close to 40,000 lives each year and avoid disability to two millions of our citizens.

The question of whether compensation should be paid where no wage loss results but on the basis of physical, social and esthetic consequences is a pertinent question which the people of each State under our system of government must answer in the light of their concept of sound public policy and economic cost, the effect of competition of local State industries with those of other States, the demands of employes and labor organizations and the knowledge derived through education as to ultimate results to industry, workers and the consumers who make up our society.

In reaching decisions we shall need the best information and guidance available. The engineer, the doctor, the lawyer, the scientist must join in the task of assembling facts and theories and applying sound reasoning in reaching a conclusion.

The Wisconsin experience is interesting because of the progress which has already been made. Before 1919 the Wisconsin compensation act provided benefits only for accidental injuries. Later a blanket provision was adopted to include all injuries and diseases growing out of, and incidental to, employment. No special procedural machinery was set up for processing claims for occupational disease. The law was amended to define "injury" as "mental or physical harm to an employe." "Time of injury" was defined as "date of the accident which caused the injury or the date when the disability from the occupational disease first occurs." One of the conditions was that an employe *at the time of his injury* must be performing service growing out of and incidental to his employment. Under these provisions the court held in silicosis cases that there must be injury during a period of actual employment, and that the injury must be *disability* such as to cause *wage loss*. This holding excluded cases in which disability resulted, not during a period of employment but after the employe had ceased service.

To do justice to these cases the law was amended to provide that in the case of disease, time of injury should be "the last day of work for the last employer whose employment caused disability." This provided a date to fix wage basis and to commence the running of the statute of limitations.

The Wisconsin act, in common with many others, contains schedules of fixed benefits. Loss of hearing in one ear called for payment of 50 weeks, and total deafness 333 1/3 weeks, which, at the maximum rate, amounted to something over $12,000. There was provision for partial deafness so that for 50 per cent deafness of both ears the allowance would be over $6,000.

A few cases were voluntarily compensated on the assumption that the schedule applied to cases of gradual loss of hearing due to noise.

Rather abruptly large numbers of controverted claims came up for decision. These presented a number of difficult questions. Was such loss of hearing an occupational disease? Could cause of loss and extent of loss be fairly determined? Was it necessary to have a "last day of work?" If so, could there be a "last day of work" when in fact employes continued at their work? Was wage loss necessary? Did the schedule apply?

The commission considered that it was its duty to interpret the law to effectuate the apparent legislative intent. It believed that the schedule, having pre-existed the occupational disease law, was read into the law when adopted and, therefore, was to be applied to occupational disease as well as to accidental injury. The legislature could easily have excluded application of schedules in categorical terms. Deafness caused the same inability to hear whether due to accident or occupational disease. Many schedule injuries do not actually result in wage loss. Was an exception to be made as to loss of hearing, although in all other types of disability schedules were to be applied?

In the test case decided by the commission, that of Wojcik vs. Green Bay Drop Forge Company, the commission concluded that if a "last day of work" was necessary it might well be considered as the last day of work before the filing of claim. The commission considered that a "token" quitting for a temporary period in order to achieve a "last day of work" would be a fatuous gesture without logical basis.

The commission found it to be established that noise can and does result in loss of hearing. Applicant worked in noise of a kind and over a sufficient period to result in loss of hearing, some of which was permanent. Claim was made within the statutory period of limitations and, therefore, was not barred.

Use of the A.M.A. table was made in determining the percentage of deafness with one variation to satisfy statutory specifications.

The commission further decided that there should be reduction in the recorded percentage of loss because of hearing loss common at the age of the employe. Under the Wisconsin statute there is a requirement that the number of weeks payable for permanent disability be reduced by 2½ per cent for each year over 50. Regardless of this provision it was established that older persons have loss of hearing greater than that found in younger persons. A deduction, therefore, was made of 7.07 per cent because of this factor. It was established that although some restoration of hearing might be anticipated, it could not amount at the most to more than 25 per cent, leaving 75 per cent as clearly due to work exposure. Reduction was accordingly made. The award was for 13.5% residual binaural loss, calling for 45.04 weeks of compensation in the sum of $1,575.46.

The compensation act provides that review of the decision of the commission as a body shall be by the Circuit Court of Dane County.

The Wojcik case was reviewed by the Circuit Court, which held that there was no "last day of work"; that before compensation could become due for an occupational disease an injured employe must have suffered disability represented by physical incapacity to work such as to cause wage loss. The court stated that schedules of fixed benefits did not apply as to occupational disease, and that it was not foreseen or anticipated that disease could produce a scheduled injury.

The court commented that no hardship would be worked on Wojcik, as he was still employed without wage loss, and that if and when his employment was terminated and wage loss resulted, he would then be entitled to compensation. In the meantime, if the legislature wished, it could change the rule. Employers could also supply safety devices to prevent or minimize exposure to noise.

The case was appealed to the Wisconsin Supreme Court, which made its decision on October 6, 1953. In that decision the court upheld the findings and order of the commission and reversed the decision of the Circuit Court.

It held that wage loss was not necessary in order to establish a claim for loss of hearing by prolonged exposure to noise; that the last day of work applied only in cases where an employe had actually quit his work, and that the commission had properly fixed the day before the filing of the application as the date for liability. (That was the approximate date of examination based upon which claim was made).

It further held that termination of employment was not a condition precedent to establishment of claim. It stated that loss of hearing was disability established by schedule within view of statute, and, therefore, such cases were compensable. Any other result the court held would be absurd and unreasonable.

Upon application for rehearing the court reiterated its decision and affirmed the commission's order.

Effective July 1, 1953, the legislature eliminated the schedule as to loss of hearing due to prolonged exposure to noise and provided for compensation for "occupational" deafness purely on a wage loss basis in sum not to exceed $3,500. In order to recover under this provision, employes must show that they were discharged or transferred due to loss of hearing or that it is inadvisable for them because of their loss to continue in their employment. That is the law now in force in Wisconsin.

The advisory committee requested the Industrial Commission to study and evolve a formula for determination of loss to be submitted to the committee for its consideration. The thought was expressed that the present AMA formula is not realistic, particularly in that it does not sufficiently measure and give credence to the factor of ability to hear and understand ordinary conversation. A medical subcommittee was appointed which has made its report.

Dr. Meyer Fox, a member of this Society, is a member of the subcommittee. He has been of great assistance to all parties and to the commission because of the profound knowledge acquired by years of study, research, examination of employes who have sustained loss of hearing, and in counseling with both industry and labor as to noise hazards and hearing loss.

The report of the subcommittee made reply to a number of questions which were submitted by the commission.

In brief, it was recommended that sound below 90 decibels as measured on a C scale of an approved sound level meter should not be considered harmful, regardless of the length of exposure. The energy per octave band determines the hazardous noise level. The inference to be drawn is that when there is noise above 90 decibels employers should be on their guard.

Losses for compensation purposes should be confined to those occurring in frequencies ordinarily used for speech conversation. Losses of hearing ability for frequency tones above 2,000 are not to be considered as constituting disability for hearing conversation. Although it was recognized that speech audiometers have considerable value, until they have been generally adopted and their use developed, pure-tone air conduction audiometric tests are to be used in evaluating hearing acuity in the readings of 500, 1,000 and 2,000 cycles. Frequencies between 250 and 8,000 cycles per second are to be used for diagnostic purposes.

The losses in the three frequencies are divided by 3 to get the average decibel loss. According to a table the loss is then converted into percentage loss. Losses averaging 16 decibels or less are not to be held to constitute hearing disability, and losses of 80 decibels and over are to constitute total deafness. An average decibel loss of 25 produces 13.3 per cent, 35 produces 40, 50 produces 55, 60 produces 71.7, and 70 produces 86.2. Each average decibel loss between 17 and 79 is ascribed a percentage of compensable hearing loss. For binaural loss the statutory formula is used, with the recommendation that for purposes of legislation the relative value of loss as between one and both ears should be as 1 to 5. Loss for presbycusis is subtracted at the rate of ½ per cent at age 50, and an additional ½ per cent for each year thereafter. Recovery of hearing ability may be expected after removal of an individual from a noisy environment. Just how much recovery will depend upon the factors of years of exposure, degrees of loss, and individual susceptibility. A first examination should be made after 48 hours removal, followed by closely spaced periodic tests. Five decibels are to be deducted from the average ratings of the 500, 1,000 and 2,000 frequencies to allow for the "recovery factor." The result will be the final permanent loss as of the time of examinations so made, except as to individuals who have been removed for six months or longer, in which case a final determination can be made upon actual readings obtained without reduction. Improvement by hearing aid is not to be allowed. Loss cannot be improved by medical or surgical treatment.

Members of the advisory committee emphasized that their recommendations cannot be recommended as final but are based upon the best scientific information now available, subject to revisions as additional information accumulates.

The Advisory Committee is continuing to study the subject with a view to evolving new proposals for legislation if considered desirable. The crucial question will be whether loss of hearing shall be compensated strictly on a wage loss basis, or whether some schedule shall again be adopted. The scope of a proposed schedule will probably depend to a considerable degree on the nature of the formula to be used.

What should the employer's position be? There can be no doubt but that employers should use all possible diligence in surveying conditions in their plants, ascertaining whether detrimental noise is present, and doing whatever is feasible to eliminate as much noise as possible. Engineers have come forth with many suggestions as to redesign, repair and maintenance of machinery and equipment. Acoustical materials are applied to advantage where effective. Segregation of noisy operations has removed the noise hazard as to some employes.

Undoubtedly the first line of defense calls for the use of ear defenders or plugs until better and more positive methods can be adopted, or in places where it may be impossible to reduce noise to a safe limit.

Labor will need to cooperate in making use of all safety devices provided. Above all, physical examinations for loss of hearing should be made promptly and at intervals. Is it possible to detect susceptibility of employes to noise? Suggestions have been made by otologists in that regard. If it can be shown that some employes are especially susceptible to noise they should either be protected or transferred to employment where the hazard of noise is not a factor. When claim is made an employe should be able to establish what loss of hearing was present at a given time and employers should, as a matter of defense, likewise be able to establish cause of loss and extent as of the beginning of employment.

Regardless of the basis of recovery, either under compensation acts or at common law, workers are rightfully going to insist that working environments are provided which will, within reasonably attainable bounds, eliminate the hazard which causes loss. As people are continuing to live to older ages, they must carry their infirmities longer. They are entitled to work in environments such as to insure retention of their faculties and senses as long as possible. The time has not yet come when codes and standards of safe practice can be written into law with sanctions for violation. That should not be done until it is certain that they can be established to be physically and economically feasible of attainment, and be accomplished upon sound engineering and safety principles.

Employers who act promptly and effectively will be able to congratulate themselves on the fact that they are carrying out the golden principle of man's humanity to man; that they are acting to preserve the precious heritage of hearing, and that their industries will not be embarrassed by claims founded on avoidable hazards.

The otologist is an indispensable member of the task force engaged in conservation of industrial hearing and in programs for determination of loss. His guidance and counsel are constantly needed by all of those who are concerned with the questions of causation, measurement and prevention of loss of hearing. It is he who has brought the subject to th front. It is he who will play a most vital role in its solutior

THE ROLE OF TRIANGULATION ROENTGENOSCOPY AS A METHOD OF GUIDANCE IN THE REMOVAL OF OPAQUE FOREIGN BODIES BEYOND BRONCHOSCOPIC VISION.*†‡

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HISTORY.

George W. Grier first devised a biplane fluoroscope for use at the Chevalier Jackson Clinic at Pittsburgh in 1910. Jackson[1] discussed the subject in his textbook, "Peroral Endoscopy," published in 1914. Grier[2] described his apparatus in a paper published in 1916. In 1932, an excellent paper on biplane fluoroscopy was published by H. K. Pancoast, E. P. Pendergrass and G. Tucker.[3] Willis F. Manges[4] in 1933 published his paper on biplane fluoroscopic aid in the removal of foreign bodies. This method had been successfully used since 1917 at the Jackson Clinic at Philadelphia.

Fluoroscopically guided bronchoscopy was fully discussed by Jackson[5] in a later textbook, "Bronchoscopy and Esophagoscopy" (1922, 1927 and 1934). Jackson and Jackson[6] demonstrated the invaluable aid of the biplane fluoroscope in "costophrenic bronchoscopy" in their article, "Pins at the Periphery of the Lung." The same authors[7,8] described in detail the subject of fluoroscopically guided bronchoscopy in two more recent textbooks. Murdock Equen[9] reported his experiences with the biplane fluoroscope in 1934. In 1936 Chevalier L. Jackson,[10] in a paper on foreign bodies, reported a case in which a tooth fragment was lodged in a small peripheral branch bronchus. The extraction of this foreign body was made possible only by expert biplane fluoroscopic guidance. In the same year, Jackson[11] and his roentgenologic co-author, Edward Chamberlain, described the biplane apparatus used in the Temple University Clinic. Chamberlain[12] added much to the improvement and refinement of the present day biplane

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† From The Charlotte Eye, Ear and Throat Hospital.

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uoroscope. In their latest textbook "Bronchoesophagology," ne Jacksons[13] covered the subject of fluoroscopic bronchoscopy in a very concise manner.

No historical discussion of this subject is complete without mentioning the newly developed stereofluoroscope. Alden H. Miller,[14] in an article published in 1951, gave credit for its development to LeRoy J. Leishman, who spent many years in perfecting the instrument. Miller and his co-workers at the Los Angeles Children's Hospital have found the stereofluoroscope the machine best suited for fluoroscopic direction of the endoscopist. This seems to be a real advance; however, the apparatus is expensive and complicated, and discussion

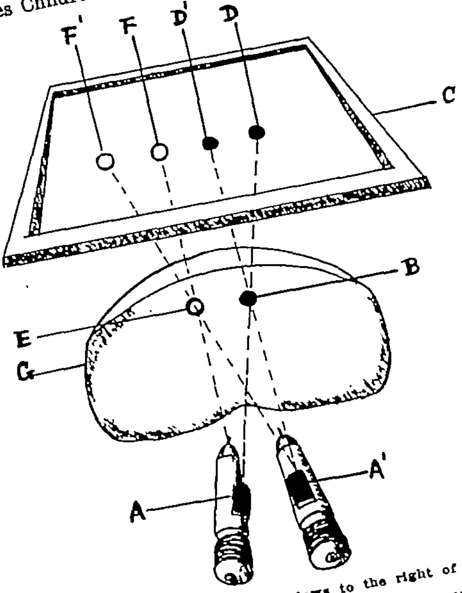

Fig. 1. Forceps or magnet shadows to the right of the foreign body shadows (wrong vertical plane).

AA^1—Roentgen Tubes. B—Foreign Body. C—Fluoroscopic Screen. DD^1—Foreign Body Shadows. E—Forceps or Magnet. FF^1—Forceps or Magnet Shadows. G—Cross Section of Human Body.

of a less costly and less complicated single screen method is pertinent.

Consequently, triangulation roentgenoscopy is reviewed in this paper. Although it has received little recognition, the method of triangulation roentgenoscopy is not new. The first article was published in 1938[15] followed by a more detailed description in 1944.[16] Clinical reports have followed.[17,18] Its usefulness has been well demonstrated in my experience.

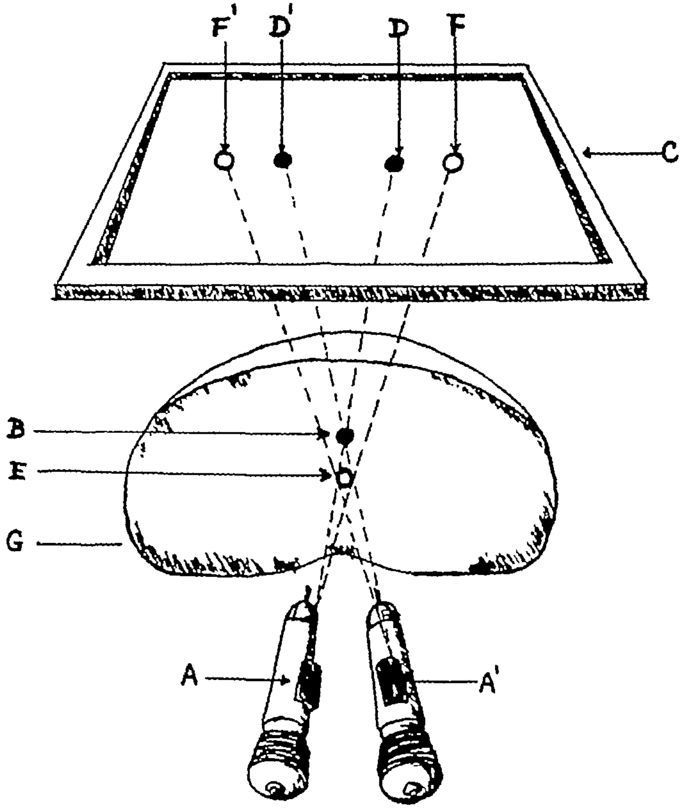

Fig. 2. Forceps or magnet introduced posterior to the foreign body. Images, therefore, farther apart than images of the foreign body.

AA¹—Roentgen Tubes. B—Foreign Body. C—Fluoroscopic Screen. DD¹—Foreign Body Shadows. E—Forceps or Magnet. FF¹—Forceps or Magnet Shadows. G—Cross Section of Human Body.

Triangulation roentgenoscopy is superior to biplane roentgenoscopy in that a single fluoroscopic screen is employed. Biplane roentgenoscopy requires constant shifting from one plane to the other. Using the single fluoroscopic screen in triangulation roentgenoscopy, all relationships of the foreign body or forceps can be clearly determined. The movement of the foreign body or instrument can be seen by the roentgenoscopist at all times in all planes. All dimensions are obtained by positions and relations on a single screen and are always visible.

THE TRIANGULATION ROENTGENOSCOPE.

The triangulation roentgenoscope consists of two tubes underneath the table on a single carriage so that the central rays of either tube may be adjusted to any desired angle (cross fire for triangulation.) It is a simple procedure to keep the amperage on each tube equalized, because each tube has its own filament control. The control panel is so wired as to permit the use of one or both tubes; thus, it does not inter-

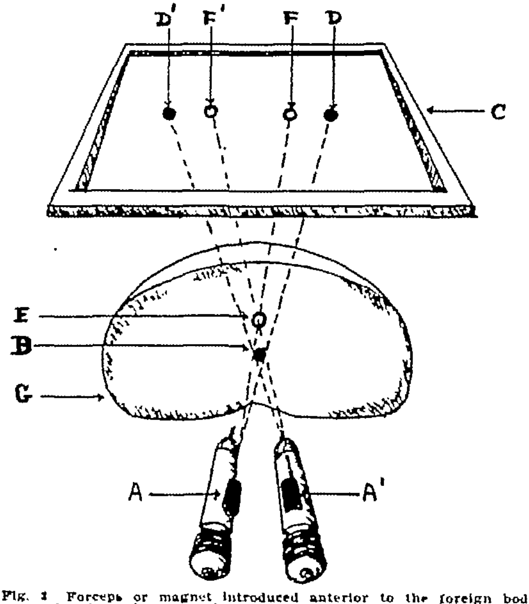

Fig. 3 Forceps or magnet introduced anterior to the foreign body. Images, therefore, closer together than images of the foreign body.

AA¹—Roentgen Tubes. B—Foreign Body. C—Fluoroscopic Screen. DD¹—Foreign Body Shadows. E—Forceps or Magnet. FF¹—Forceps or Magnet Shadows. G—Cross Section of Human Body.

fere with conventional roentgenoscopy. The shutter opening must be of special design, using a larger aperture.

Since both tubes are activated at the same time, their central rays intersect, and there are two shadows of the foreign body on the fluoroscopic screen instead of one. Any other opaque object which comes into the field, such as a pair of forceps or a magnet, will also produce two shadows on the fluoroscopic screen. If the forceps shadows are to the right or left of the foreign body shadows, but in the same horizontal plane, it is obvious that the forceps are in the wrong vertical plane (see Fig. 1). If the forceps or magnet are introduced into a plane posterior to the foreign body, the images of the forceps or magnet will be farther apart than the images

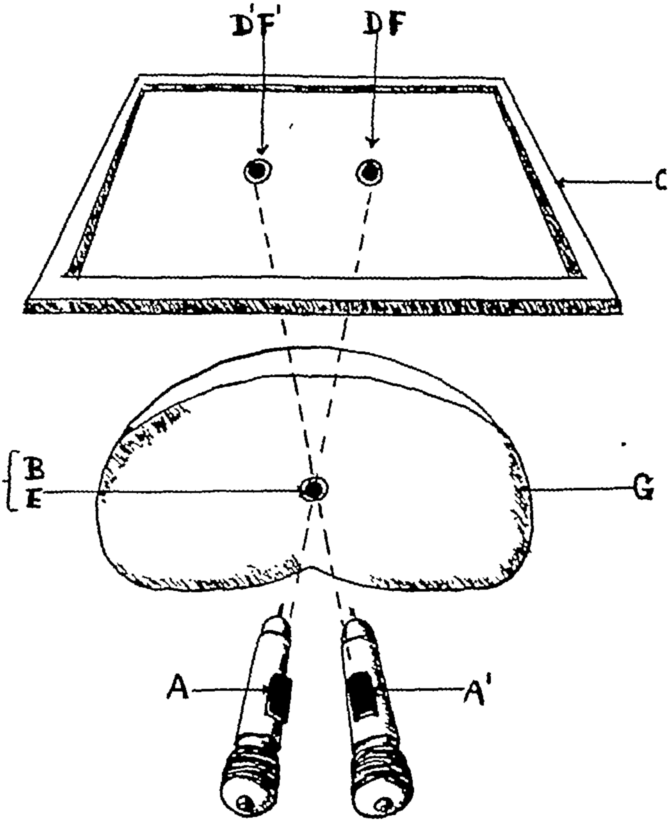

Fig. 4. Forceps or magnet introduced into the same plane as the foreign body. Images, therefore, coincide.
AA^1—Roentgen Tubes. B—Foreign Body. C—Fluoroscopic Screen. DD^1—Foreign Body Shadows. E—Forceps or Magnet. FF^1—Forceps or Magnet Shadows. G—Cross Section of Human Body.

of the foreign body, because the triangles formed by the intersecting rays to the foreign body and magnet are unequal (see Fig. 2). Introduction of the forceps or magnet into a plane anterior to the foreign body will conversely show the forceps or magnet shadows closer together than those of the foreign body (see Fig. 3). When the forceps or magnet are introduced into the same plane as the foreign body, the distance between the foreign body shadows and the forceps or magnet shadows will be the same (see Fig. 4). This is true because the triangles formed by the intersecting rays to the foreign body and the forceps or magnet are identical in size.

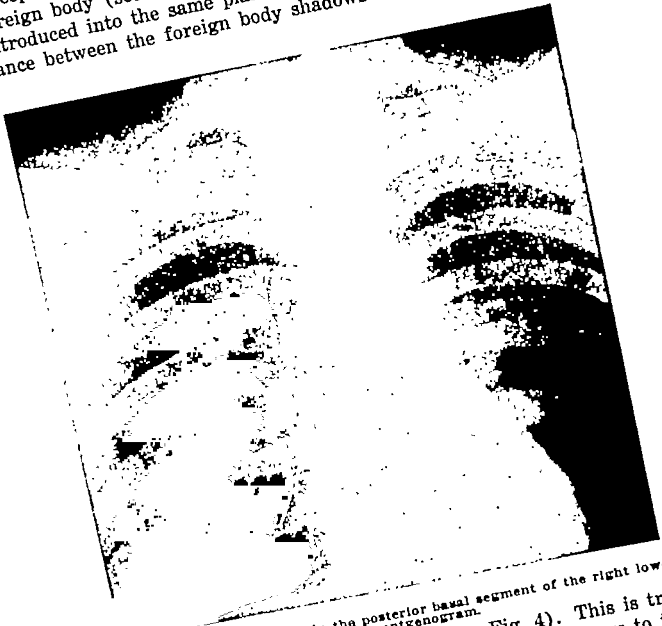

Fig. 5. Escutcheon nail in the posterior basal segment of the right lower lobe bronchus. Postero-anterior roentgenogram.

REPORT OF ILLUSTRATIVE CASES.

The following cases illustrate the practical application of combined triangulation roentgenoscopy and bronchoscopy. The successful solutions to these problems resulted only from the undivided cooperation between the bronchoscopist and the roentgenoscopist.

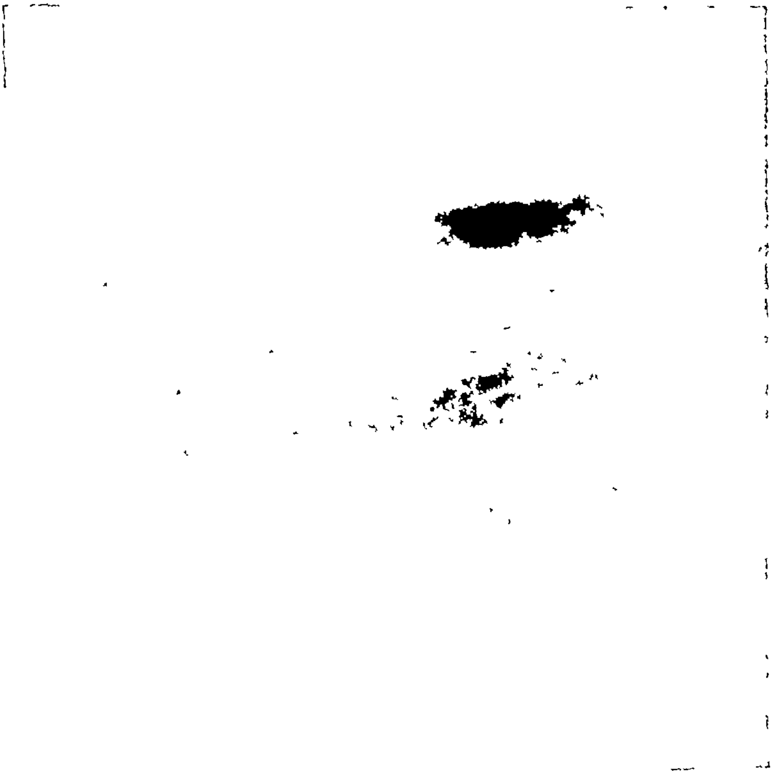

Fig. 7. Magnet shadows to the right of the nail shadows.

Case 1. J. C. M., a white male, aged 37, was referred on November 2 1951. While attempting to weatherstrip his house the patient had accidentally aspirated a small escutcheon nail. Roentgenograms revealed the foreign body to be a small nail located in the right lung, probably beyond endosopic vision (see Fig. 5). A duplicate of the foreign body proved to be a magnetic escutcheon nail measuring 1.7 cms. in length.

On November 3, 1951, bronchoscopy was done with topical anesthesia and the nail found to be beyond endoscopic vision. With the aid of triangulation roentgenoscopy, an Equen[19,20] bronchoscopic magnet (3 mm. diameter attached to a woven stem) was inserted into the various orifices of both the middle and lower lobe bronchi by the trial and error method. Figure 7 illustrates that the magnet was to the right of the nail. Figure

8 illustrates that the magnet was to the left of the foreign body. Work was discontinued because it was felt that the patient had had enough manipulation for one day.

On November 5, 1951, another attempt to make contact with the nail was unsuccessful.

On November 15, 1951, the entire procedure was repeated under local anesthesia. A 7x40 standard Jackson bronchoscope was introduced without the use of a laryngoscope. The subdivisions of the lower lobe were exposed. The Hollinger[illegible] vertebrated tip magnet was passed into the superior segment, the anterior basal segment, the lateral basal segment and the medial basal segment.[illegible] In none of these was the magnet near the foreign body. The magnet was then passed into the posterior basal segment, and the roentgenoscopist said it was nearer the foreign body than heretofore.

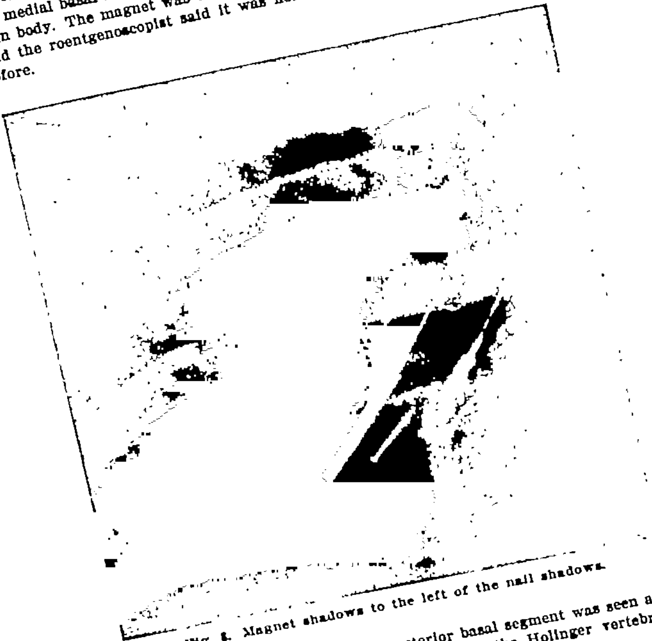

Fig. 8. Magnet shadows to the left of the nail shadows.

A small segmental orifice of the posterior basal segment was seen anteriorly. This would admit neither forceps nor the Hollinger vertebrated tip magnet. After some difficulty, the 3 mm. Equen Alnico magnet was passed into this subdivision. Advancing the magnet, contact was established (see Fig. 9). Upon withdrawing the magnet, the nail moved with it (see Fig. 10). The magnet and attached nail were then extracted through the bronchoscope. The total operating time was approximately 20 minutes The patient was dismissed the next day and had no further difficulty

Case 2. H. K., a white male, aged 10 months, was referred on February 20, 1952 On February 18, 1952, the baby picked a pin out of a chair and put it in his mouth The grandmother immediately put her finger in the baby's mouth but only succeeded in pushing the pin downward. At this point the baby aspirated it and began to cough violently. The patient was taken to a university medical school center, where it was discovered that the foreign body was in the left lower lobe of the lung. Bronchoscopy there revealed no evidence of the foreign body. Obviously, the pin was beyond endoscopic vision. The baby was then referred for further treatment because of a lack of necessary fluoroscopic equipment. Roentgenograms (see Fig. 11) revealed a straight pin, head down, in the left lower lobe, probably the posterior basal segment.

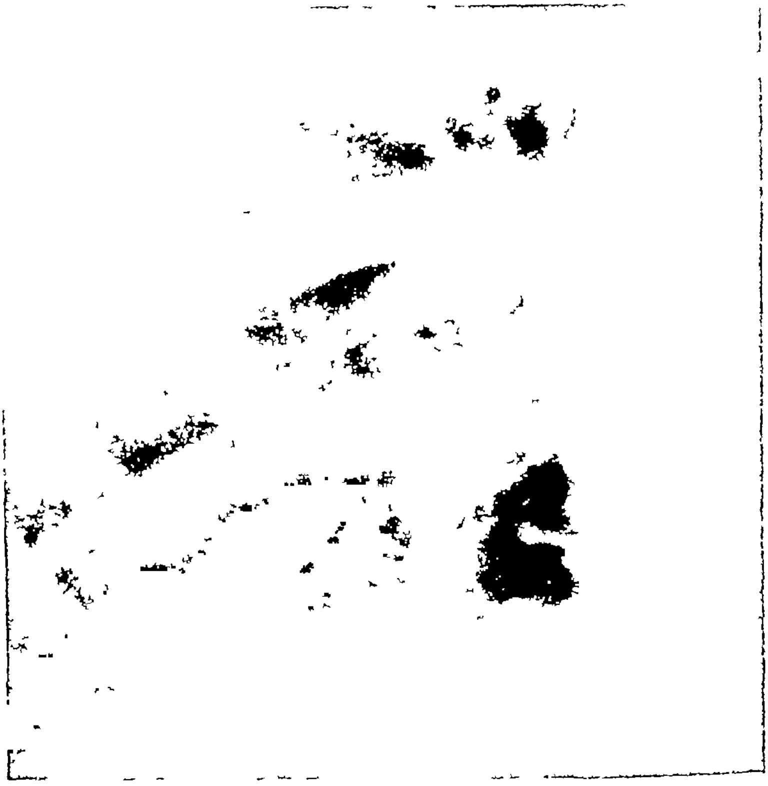

Fig 9 Magnet in the proper plane because the distance between the nail shadows and the magnet shadows is the same

Combined bronchoscopy and triangulation fluoroscopy was done on February 20, 1952, without anesthesia. A 3½ mm. Jackson bronchoscope was introduced through a laryngoscope. The bronchoscope was advanced into the left mainstem bronchus. After exposing the subdivisions of the left lower lobe, 1 cc. of adrenalin and saline was instilled and the excess taken out with a small bronchoscopic suction tube. Delicate side curved forceps were advanced into the various subdivisions of the left lower lobe. Using the trial and error method the forceps were first introduced into the anterior segment, and they were too far to the left. Advancing the forceps into the lateral segment they were too far anteriorly. Contact was then established in a small subdivision of the posterior basal segment. The forceps were advanced to the point of the pin, open and closed under guidance of the roentgenoscopist. The foreign body was engaged and extracted without difficulty. The total operating time was approximately eight minutes. The patient was dismissed from the hospital on February 21, 1952.

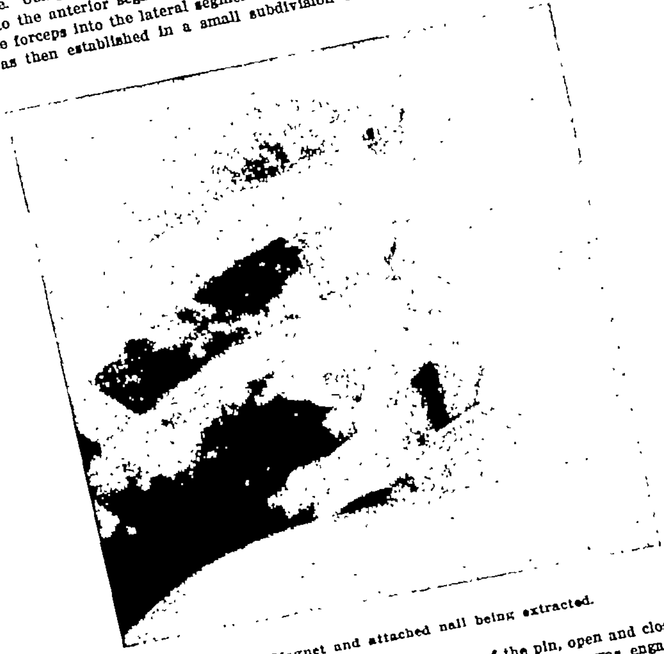

Fig. 10. Magnet and attached nail being extracted.

Case 3. C. H., a white male, aged 17, was first seen on April 13, 1952. The patient aspirated a tack on April 10, 1952, and was bronchoscoped elsewhere, but no foreign body found. Evidently, the foreign body was beyond bronchoscopic vision.

A postero-anterior roentgenogram (see Fig. 13) revealed a shadow of a tack in the lower right lobe of the lung immediately adjacent to the right heart border just beyond the crest of the diaphragm In the lateral view, the tack shadow was overlying the vertebral column.

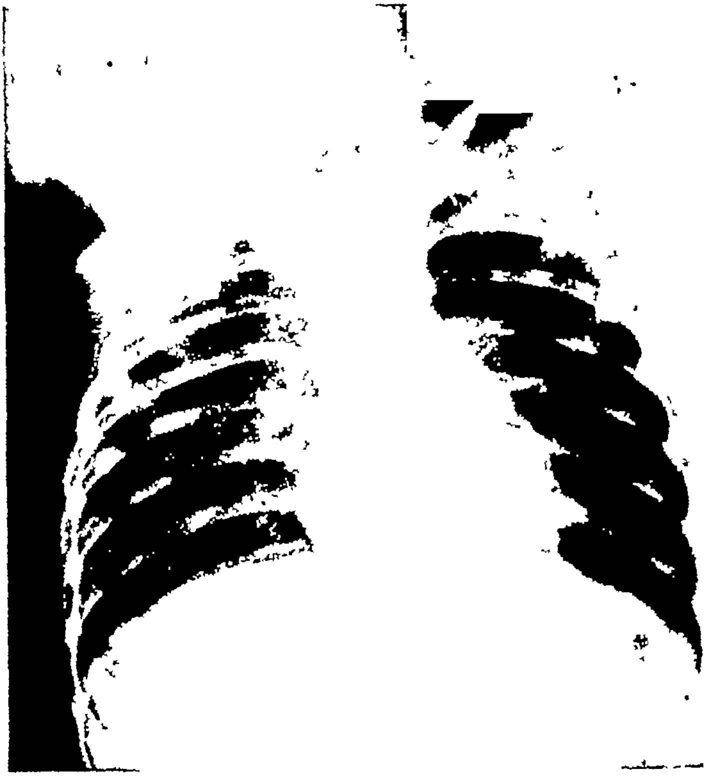

Fig 11 Postero-anterior roentgenogram showing straight pin, head down, in the posterior basal segment of the left lower lobe

Bronchoscopy, using triangulation roentgenoscopy, was performed under local anesthesia A full lumen No 7 Jackson bronchoscope was introduced without a laryngoscope, and the various subdivisions of the lower right lobe were brought into view By searching the branches, the small Equen Alnico bronchoscopic magnet was finally inserted into the correct subdivision This proved to be the posterior basal segment. Contact was established between the tack and the magnet, and the foreign body was gently withdrawn through the bronchoscope. The total operating time was about ten minutes. The patient was dismissed the following day.

Case 4. R. L. W., a white male, aged 52, aspirated a dental inlay on September 29, 1952, while in the dentist's chair. The patient began to cough, and the referring dentist was alert enough to have a roentgenogram made. This showed the foreign body to be in the lung.

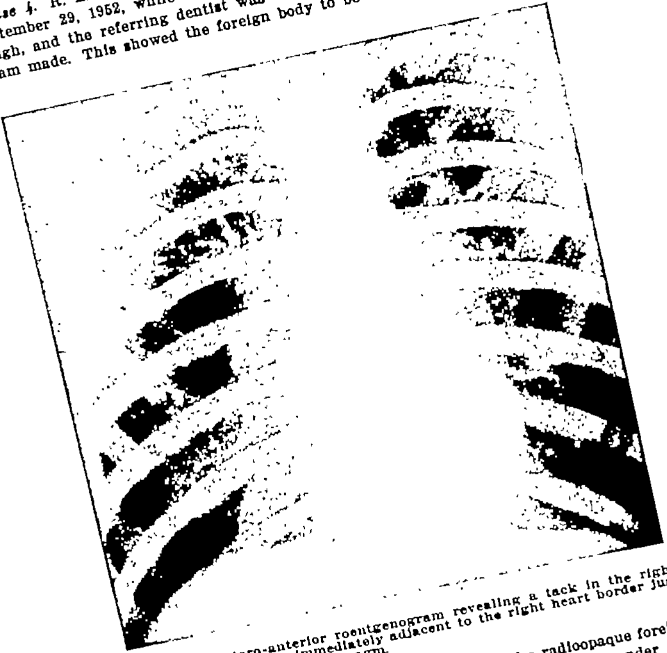

Fig. 12. Postero-anterior roentgenogram revealing a tack in the right lower lobe of the lung immediately adjacent to the right heart border just beyond the crest of the diaphragm.

Roentgenograms made on October 1, 1952, revealed a radioopaque foreign body in the right lower lobe (see Fig. 15). Bronchoscopy, under triangulation roentgenoscopic guidance, was performed on October 1, 1952. After preliminary medication, the airway was prepared with topical cocaine anesthesia. A 7 mm. Jackson bronchoscope was passed without the use of a spatula. The foreign body, as expected could not be visualized. Delicate 50 cm. side curved forceps were introduced into the lateral segmental bronchus of the right lower lobe. It was obvious that the forceps were in the wrong bronchus. The forceps were then introduced into the posterior basal segment. Contact was certain. After some manipulation, the foreign body was seized and extracted. The total operating time was approximately 12 minutes. The patient was dismissed the following morning and had no further difficulty.

Case 5. V. T., a white female, aged 8, aspirated a straight pin on August 14, 1952. Two previous bronchoscopies performed elsewhere were unsuccessful. Roentgenograms (see Fig. 17) revealed a straight pin in the right lower lobe posteriorly. Bronchoscopy, with triangulation roentgenoscopy, was carried out on August 19, 1952, without anesthesia except for preliminary sedation. A 5x30 Jesberg bronchoscope was introduced. The right lateral segmental bronchus was first located, and a small pair of side curved forceps introduced. It was seen that the forceps were below the level of the pin but approximately in the correct vertical plane. The forceps were then withdrawn, and by shifting the position of the head

Fig. 15. Postero-anterior roentgenogram revealing a dental inlay in the right lower lobe.

together with further manipulation, contact was established in the anterior segmental bronchus. The point of the pin was seized and the foreign body extracted without difficulty. The total operating time was approximately five minutes. The following day, August 20, 1952, the patient was dismissed from the hospital in good condition.

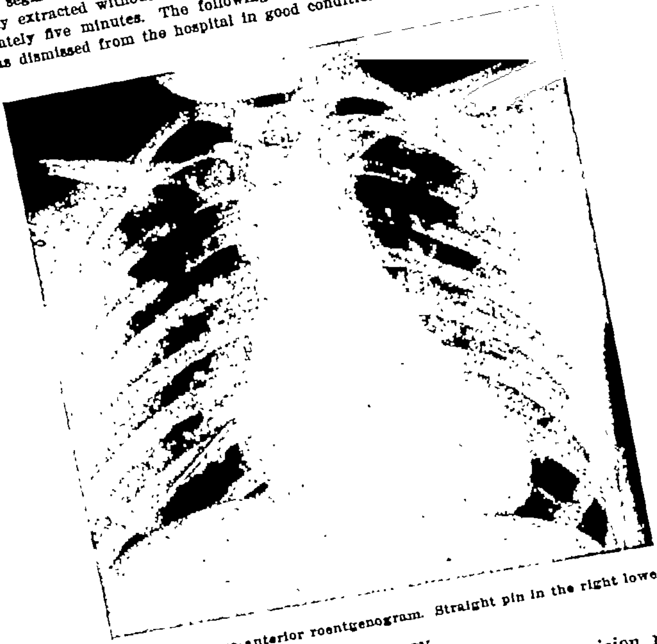

Fig. 17. Postero-anterior roentgenogram. Straight pin in the right lower lobe.

SUMMARY.

An opaque foreign body beyond bronchoscopic vision requires fluoroscopic direction. Triangulation roentgenoscopy offers practical and accurate guidance. The single screen of such apparatus is a valuable simplification of biplane fluoroscopy. The apparatus required is less complicated and much less expensive than the stereofluoroscope; therefore, a description of the mechanics involved has been presented. Case reports were also recapitulated illustrating the successful use of this method.

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A HALF CENTURY OF TRIOLOGICAL TRANSACTIONS.*

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Brookline, Mass.

To compress and encapsulate within my allotted time the content of the writings of the Triological Transactions during the first 50 years of publication would seem as difficult a task as the inscription of the Lord's prayer on the head of a pin. This otolaryngological literary output encompasses some 2200 papers totaling over 25,000 pages and 10,000,000 words, of which I must at once plead guilty to not having read every one. I am very conscious of my obligation in this survey to adhere to the oft heard injunction of one of otolaryngology's most distinguished teachers to "boil it down" and I can only hope that, like Vermont maple syrup, the remaining extract will have some body and flavor.

The Transactions are in a sense a monument to all those who, no longer here, have by their labors done so much to promote the development of this specialty and to advance its growth and achievements. They are, moreover, a history of men who have continually engaged in a ceaseless quest for the answer to some problem and in the often frustrating pursuit of an idea, sometimes up a blind alley, sometimes to a rewarding pot of gold at the rainbow's end. Shakespeare might well have had the physician in mind when he said

> "Oft expectation fails and most often
> There where most it promises
> And oft it hits where hope is coldest
> And despair most fits."—*All's Well that Ends Well.*

Many of the pioneers in otolaryngology entered it through the door of general practice and so were far more accustomed to view the patient as a whole than is the modern specialist who is ever exposed to the temptation to see only what is vis-

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*Read at the 58th Annual Meeting of the American Laryngological, Rhinological, and Otological Society, Inc., Boston, Mass., May 27, 1954

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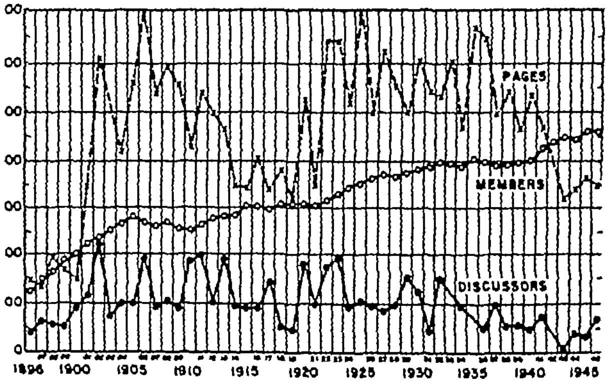
ible through the hole in his head mirror. Prior to 1900 typhoid fever, diphtheria, syphilis and tuberculosis were an every day experience, and their otolaryngological complications were wisely considered as only a part of a more widespread disease.

May I first show you on a slide the over-all picture of the growth of the *Transactions* over the first fifty years of their existence (see Fig. 1). You will note the diminished productivity occasioned by two world wars, in both of which the members of this Society served with such distinction. Growth is a hall mark of any successful project, of which a comparison of the size of the first volume and that of 1936 is striking evidence (see Fig. 2).



(Fig. 1. Graphs showing the yearly changes in size of the volumes, membership and discussions for the first half-century of the Transactions).

A few simple statistics will, I hope, not prove tedious. Of the 2,200 odd papers some 500 (25%) have dealt with nasal disease, over half of these with some aspect of sinusitis. There are some 400 papers on the throat, of which 55 per cent are devoted to the larynx. Over 500 papers deal with some phase of otology, more than half of them being concerned with acute otitic infections and their complications. One hundred and thirty papers relate to the varied aspects of endoscopy, and it is this subject, which, despite pressing competition from the American Broncho-Esophagological Society, founded in 1917, leads all others in the breakdown into single topics of discussion. In this race for popularity, endoscopy is closely pursued by the 115 papers on the complications of acute otitic infection with the hundred articles on anatomical, histological and pathological research breaking the tape neck and neck with an almost equal number on the ethmoid and spenoid sinuses, while the humble tonsil is scarcely a step behind them.

While the *Transactions* do not by any means reflect all the changes and progress made during the past fifty years, they nevertheless afford a fairly reliable index of the specialty's major developments, since among the contributors are numbered the foremost workers in otolaryngological history. Specific dates assigned to certain surgical innovations or therapeutic developments are not always those at which these events first occurred but rather the years when first encountered in the *Transactions*.

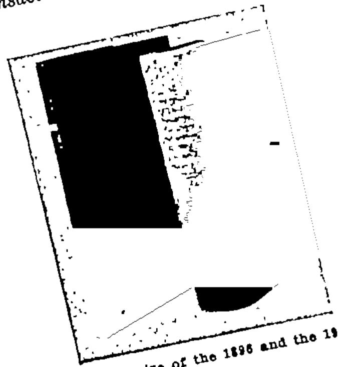


Fig. 2

(Fig. 2. Comparative size of the 1896 and the 1926 volumes).

At the time of the Society's first meeting in New York in 1896 it was being said by some, let us hope facetiously, that otolaryngology was a specialty of "sprays and spurs, of snares and syringes." If so, it has come a long way in the past 50 years. True, the specialist's chief armamentarium consisted of his aural and nasal speculae, his atomizer and his Politzer bag. It was a day when the term laryngoscopy signified only the indirect use of the laryngeal mirror whereby with a dexterity seldom matched today, the laryngologist undertook the therapy of laryngeal disease, most often in his office or in the patient's home. Faced with a foreign body in a child's bronchus, he had nothing to offer beyond a tracheotomy and the hope that with God's providence the tickling of the trachea with a feather would induce sufficiently violent coughing to expel the intruder. Only by hearsay had there come rumors from a famous German clinic of a tube to be passed directly into the trachea, thus heralding the development of the whole field of endoscopy.

Leeches were considered a feasible means of attempting to forestall the development of acute mastoiditis.

In 1900 several of the most eminent members of this Society expressed themselves in favor of adenoidectomy in children without any anesthesia even though necessitating a piecemeal operation at several sittings. It was said any child, if held by two strong adults, could be satisfactorily restrained. All to frequent fatalities attributable to general anesthesia led parents to elect this seemingly brutal procedure as the lesser of two evils.

At the turn of the century little besides inflation and bouginage could be offered the chronically deafened patient and ossiculectomy for chronic otitic suppuration was in such vogue that one author could personally report a recent series of over 200 cases. Such complications of otitic sepsis as meningitis and brain abscess were viewed with almost universal acceptance of a fatal outcome.

As concisely as possible I should like now to trace the progress and achievements which have occurred during the half century in certain major fields of otolaryngology. Such a survey must necessarily omit many details in the effort to present a bird's eye view of the picture without exhausting your patience.

TONSILLECTOMY.

Throughout the years the operation of tonsillectomy has probably been done more frequently, oftentimes more inexpertly, and in the long run, with more benefit to the patient than any other surgical procedure in history. There are almost *100* papers on some phase *of the tonsil* problem, *two-thirds of them written before 1926.* In the last 20 years there have been only 17, indicative of the feeling that the whole subject has been worn rather thin.

The earliest tonsil operation consisted of crude multiple attempts to open by curettage, and so to facilitate cauterization, of the tonsil crypts. Not till 1901 was tonsillotomy in vogue. The hemorrhage and recurrent infection led to the substitution of the conventional capsular enucleation in 1907, at first by simple finger dissection and later with the cold wire snare, by which time we already read of protests against excessive and unnecessary operating.

In 1912 there came on the scene the innovation of the guillotine technique, which rapidly gained popularity, and which in a variety of modifications shares with wire snare dissection the bulk of tonsil surgery.

The unhappy complication of lung abscess following tonsillectomy created interest, about 1926, in the merits of the prone and the sitting position, and in 1931 reports of sudden operative death lead to suspicion of enlargement of the thymus gland as the culprit, and the advocacy of routine X-ray examination and preventive radiation therapy.

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Considering the frequency of the operation, adenoidectomy has been a subject of surprisingly infrequent attention in the *Transactions* with a total of only 19 papers in the fifty years; but four of them in the last ten years. The procedure at the turn of the century was by modern standards a crude affair, performed often without anesthesia, at two or more sittings, frequently in the home or the office, with incomplete removal followed by sometimes alarming hemorrhage and with instruments now to be found only in a hospital museum. Within ten years, however, efforts were being made to operate under direct inspection by elevation of the soft palate and thereby to achieve far more complete removal of all visible lymphoid tissue, a method destined to be revived and in a sense rediscovered 40 years later.

The concept of the probable influence of adenoid pathology on disease of the middle ear dates back to the very earliest days of the Society and the relationship of adenoid hypertrophy to mouth breathing, and the general hygiene of the patient was well understood 50 years ago.

ATROPHIC RHINITIS.

The stubborn problem of atrophic rhinitis and efforts to deal with it date back to 1907. Attack via nasal sinus surgery, by investigation of the bacteriological aspects and attempts to discover a specific causal organism led, in 1916, to a wave of enthusiasm for polyvalent vaccines. Surgical measures to increase the width of the septum bore meager fruit, and only within the past few years have more encouraging results appeared to follow the application of chemotherapy. There is a widespread feeling that, apart from the merits of any form of therapy, the incidence of this disease is decreasing, only nine papers having been published during the first 50 years of the *Transactions*.

SEPTUM.

Second only to the tonsil, the nasal septum has been a subject of interest from the very inception of Triological meetings. One may be pardoned a tolerant smile as he reads a description of the earliest procedure for the correction of septal deflections and deformities. It consisted essentially of cross hatching and sawing of the deviated cartilage to break up its resilience in combination with a forceful fracturing of the maxillary ridge to further mobilize the whole septum. Retention of the partition in its new and desired position was achieved by some form of retaining splint, usually of hard rubber, which was left in the nasal passages for from 10 to 30 days, with such discomfort to the patient as can well be imagined. In addition, rhinologists of the highest standing recommended, as further assurance of future patency of the airways, the complete removal of the inferior turbinate on the concave side. In later years this technique was to be referred to in one presidential address as "brutal and fantastic," an indication that a constantly changing viewpoint is inherent in all medical progress. Hemorrhage, inadequate anesthesia and all too frequent septal abscesses made this operation increasingly unsatisfactory until 1903, when the introduction of the swivel knife changed the whole picture and afforded a rare instance in which a single instrument has been largely responsible for a complete change in operative technique. Thus was born the modern submucous resection, which in essential details has remained a standard procedure for over 50 years. Prolonged and discomforting splints and packing have become a thing of the past, and a more wholesome respect for the inferior turbinate is now universal.

Over half the papers dealing with septal operations are to be found in the first ten volumes of the *Transactions*. Those in later years have dealt largely with operative indications and the relationship of septal deformities to the broader field of rhinoplasty, in which the operation of today is so often incorporated.

NASAL SINUSES.

From the anatomical, histological, cytological and pathological viewpoint, more time and study has been developed by the membership of this Society to the nasal sinuses than to any other structure coming within the scope of otolaryngology. No less than 94 papers have dealt with the maxillary antrum, 96 with the sphenoid and ethmoid sinuses and 66 with the frontal sinuses. The many writings on the antrum illustrate with crystal clarity that rhinologists, like the boys and girls in Iolanthe, "are all either a little radical or else a little conservative."

Throughout the whole half century *Triological* rhinologists have been about equally divided between those who inclined to the intranasal approach to infections of the antrum and those who preferred some more radical form of surgery. One might even go further and subdivide each major group into conservative radicals and radical conservatives. At the turn of the century the only approach to maxillary sinus infection was through an open tooth socket through which irrigations were carried out, and the patency of which was maintained for weeks or months by means of some sort of obturator held in place by an attachment to an adjacent tooth. Even after the adoption of the canine fossa approach attempts were made with obturators to maintain a patent window through which the condition of the lining membrane could be observed. and via which curettage could be repeatedly undertaken. Indeed we read of one patient who in 1903 washed out his own antrum through this route every time he washed his face. Not till 1906 was the intranasal window added, with or without removal of the inferior turbinate, thus laying the foundation for the classical Caldwell-Luc operation. Disagreement as to the merits of removal of the lining membrane appear frequently in later publications, particularly with respect to the relationship of antral pathology to nasal allergy.

Meanwhile during the self-same period proponents of intranasal surgery for maxillary sinusitis were claiming equally good results. The application of such conservatism to sinus infections in children was widely popularized around 1930, and remarkable results were reported in restoring to health patients with a wide range of systemic complaints. The merits of various forms of post-operative treatment continued to occasion lively debate. To irrigate or not to irrigate, to pack or not to pack and for how long, the merits of this or that instillation, paste or other medication, all illustrate the healthy disagreement and individuality of opinion which has always characterized the writings of members of this Society.

ETHMOID AND SPHENOID SINUSES.

In like manner interest in the ethmoid and sphenoid sinuses, from a surgical standpoint, has been divided between those who primarily favored only an intranasal operation and those who considered an external approach to give better access to the site of pathology, particularly in the face of possible orbital or intracranial complications. Some of the so-called intranasal procedures seem to us today to have more nearly deserved the designation "radical," as witness in 1909 the reported *en bloc* removal of the entire ethmoidal labyrinth and middle turbinate in a single specimen. When critical colleagues termed this procedure "dangerous, brutal and unnecessary," its proponent denied the allegation of brutality, "so long as the patient didn't see it."

A high preponderance of papers on ethmoidal and spenoidal sinusitis deal with its relationship to disorders of the eye, particularly so-called retrobulbar neuritis. A causal connection between these sinuses and sudden loss of vision was debated pro and con year after year, and the wisdom of a surgical attack on the sinuses forms the basis of a large number of papers between 1910 and 1930. As so often happens with a controversial medical concept, the pendulum by 1938 had swung away from the earlier point of view, and in a series of 225 cases of retrobulbar neuritis at the Mayo Clinic only one was considered to be due to associated pathology of the nasal sinuses.

A similar reversal of opinion has taken place regarding the assumed role of the sphenopalatine ganglion as the cause of various forms of headache and neuralgia. In 1913 there was wide acceptance of this concept, but by 1938 existence of sphenopalatine neuralgia was being denied, and its symptoms ascribed to irritation of sensory branches of the Vth nerve.

In 1928 the valuable diagnostic and therapeutic innovation designated as "displacement" made its first appearance, and the ensuing 25 years have served still more firmly to confirm its merits and value.

FRONTAL SINUS.

It is around the most effective treatment of frontal sinus pathology that the most persistently varied point of view has constantly centered in *Transaction* papers. The merits of the external as against the intranasal approach, of a wide range of methods of avoiding deformity, of maintaining the patency, by in-dwelling gold or rubber tubes, of the nasofrontal duct, of variations in post-operative treatment, all have been the subject of continuous interest and report.

In all rhinology no operations have been the subject of such controversy and debate as those advocated for eradication of frontal sinus infections, and despite the untiring work of 50 years none has on the whole been more unsatisfactory or failed more often to achieve a standardization upon which there is even approximately general agreement today. Between the years 1930-40 the distressing osteomyelitic extension of infection of the frontal sinus led to a considerable number of papers concerned with the pathology of this condition and the surgical procedures, conservative and radical, best adapted to dealing with it. Here, happily, chemotherapy has proven of inestimable benefit, as in so many other forms of serious otolaryngological infections.

MALIGNANT DISEASE.

Paper dealing with malignant disease of the nose, throat, sinuses and to a far lesser extent, the ear, appear at the very outset of the *Transactions*. In reading of the efforts to cope successfully with cancer of the tonsil, sarcoma of the nasopharynx and the various types of malignant disease of the sinuses, one is moved to wonder at the surgical courage with which such major pathology was handled. The mortality was high and the modern aids of transfusion, diathermy, and chemotherapy were unavailable in that day. Problems of recurrence, sequestration, prolonged hospitalization and low patient, and I suspect often physician, morale were ever present. Nevertheless, radical excisions of the superior maxilla, the transantral approach to juvenile fibroma of the nasopharynx and orbital exenterations were by no means uncommon at the turn of the century. It was about 1910 that hope dawned that radiation might hold the key to this distressing problem, and perhaps eventually serve as a replacement for dangerous and mutilating surgery. Much was written on this subject between 1920 and 1935. Authors alternated between elation and despair as to the respective merits of radiation and surgery, adopting first one alone, then the other, and finally advocating a combination of both. Even today this debate has not reached a final conclusion, although the future looks continually brighter as increasingly better radiation techniques become available. The advent in 1926 of surgical diathermy has done much to facilitate all surgical procedures involved in the eradication of malignant disease and to a large extent has eliminated the serious hemorrhage, which formerly constituted such a formidable operative hazard.

As a somewhat specialized form of maligancy, cancer of the larynx has received a steadily increasing amount of attention with the passing years. Already by 1897 total laryngectomy, which we are apt to think of as a rather modern procedure, was well established, but it carried a high mortality of over 70 per cent, and recurrence occurred in three out of five cases. Even as late as 1929 it was felt by some that "complete removal of the larynx is such a formidable task, the risks are so great and the later condition of the patient so pathetic that a conscientious doctor hesitates to advise or perform it. There is no more pitiable object than a person without a larynx. Patients should be allowed to die peacefully and not be subjected to a disgusting and revolting operation."

Fortunately for the future status of laryngectomy others took a more optimistic point of view, and increasingly better results, both surgical and phonetic, lent encouragement to continuation of the operation. Already in 1904 a teamster is said to have developed a sufficiently powerful esophageal voice as to be able to cluck to his team of horses 150 feet away. By 1925 the advantages of early diagnosis were being reflected in the increasingly successful application of laryngofissure to laryngeal malignancy. Improved methods of anesthesia, the use of surgical diathermy and the application of post-operative radiation were bringing a marked reduction in mortality and recurrence. By 1933 the technique of radiation as exemplified by Coutard gave promise that eventually it might wholly supplant removal of any portion of the larynx, a hope admittedly not yet fully realized today.

One is inclined to regard supplementary neck dissection as an adjunct to laryngectomy as a quite modern procedure but as early as 1904 this method of assurance against late recurrence was well established. During the ensuing 20 years we read of frequent appeals that conventional laryngology be expanded to include the entire head, and even the thyroid gland, such that in 1928 it was even proposed to change the Society's name to that of The American Society of Head Surgeons. So the pendulum swings back and forth, and today's apparent innovations prove to be only revivals of objectives hoped for in days gone by.

LARYNGOSCOPY, BRONCHOSCOPY, ESOPHAGOSCOPY.

The half century summary of the subject of direct visual inspection of the larynx, bronchi and esophagus shows a total of 131 papers, of which but two appeared in the first ten years of the *Transactions.* Until 1905 the larynx could be visualized only indirectly with the mirror, and removal of foreign bodies in the airways was effected almost exclusively by tracheotomy and the blind introduction of some form of crude grasping instrument. In that year, stimulated by reports of its successful use in Germany, numerous members of the Society began their efforts toward utilization of direct endoscopy, which by 1909 was well launched toward an increasingly wider adoption by laryngologists throughout the country.

The *Transactions* of 1910-1920 contain paper after paper recording the successful removal of a wide range of foreign bodies from the air and food passages, a trend which was to culminate in 1923 in the report from one outstanding clinic of a series of over 1,100 foreign bodies. In addition endoscopic methods came more and more to be applied to such conditions as esophageal strictures and tumors and to the diagnosis and treatment of such medical conditions as bronchiectasis, lung abscess and pulmonary cancer. The utilization in 1925 of various types of opaque and contrast media for delineation of the tracheobronchial tree initiated a great advance in the progress toward the solution of many problems of pulmonary pathology.

By 1931 even pulmonary tuberculosis was coming to be considered amenable to bronchoscopic therapy. The constant devising of new and ever more ingenious instruments, forged frequently in the laryngologist's own machine shop, better methods of anesthesia and the further perfection of roentgenological assistance to the endoscopist have seen this special field make probably the most spectacular progress since its humble beginnings than has any other branch of otolaryngology.

LARYNGEAL STENOSIS.

Long before the advent of direct laryngoscopy, thyrotomy had been in vogue as a method of reaching various obstructive lesions inside the larynx, chiefly those complicating typhoid fever, diphtheria, infectious laryngitis. Steady technical improvements involving skin grafting, prolonged intubation and in 1933 the innovation of expanding core moulds have all brought progressively better end results. Laryngeal stenosis has been the subject of only two papers in the last ten years, probably because the originally frequent causes of such stenosis have been more and more eliminated, as preventive medicine exerts its salutory influence and becomes the real objective of all medical practice.

PAPILLOMA.

Laryngeal papilloma has proven over the years to be one of the laryngologist's most stubborn problems. From the days when attempts were made even in young children to remove these growths indirectly, the exact etiology and particularly the mechanism of local recurrence remain as much a mystery as ever. No medical or surgical procedure, whether by the local application of such medications as alcohol, formaldehyde, of podophyllin, the use of X-ray or radium, or tracheotomy, or thyrotomy, has yielded consistently satisfactory results, and superficial and repetitive removal remains today the procedure of choice. From the fact that between 1914 and 1943 not a single Triological paper deals with papilloma, we must conclude that here is a rare instance of at least the temporary abandonment of a so far futile quest.

Time permits only mention of papers on a wide range as laryngeal subjects such as vocal nodules, benign tumors, polyps, etc., and various forms of vocal cord paralyses. In 1900 tracheotomy was the only procedure available to a patient with bilateral abductor paresis. Through the disappointing transitional stages of excision of the cords, submucous cordectomy in 1937 and arytenoidectomy in 1943, steady progress has been made towards an increasingly satisfactory answer to this problem.

ALLERGY.

Few rhinological conditions have been subjected to more and more thorough study and investigation than that known today as nasal allergy. In 1896 the symptom known as rhinedema or nasal hydrorrhea was ascribed to such a varied etiology as protein sensitivity, excess uric acid in the blood, sinus infection and the reflex irritative effect on the nasal mucosa of products of tissue metabolism. Treatments by injections of fluid extract of ragweed called liquor ambrosia were the forerunner of our modern methods of desensitization.

A wave of enthusiasm for sinus surgery waxed and waned between 1920 and 1930, when it was hotly debated as to whether allergic manifestations resulted from a diseased sinus mucosa or whether the latter was a secondary complication of an underlying allergy.

By 1926 it became apparent that only through the combined viewpoint of the rhinologist and the allergist could an even approximately satisfactory solution be arrived at. For a brief period around 1936 there entered the picture the procedure of zinc ionization, involving a complicated and expensive machine, equipped with intriguing dials and gauges designed to send an electrical current through a rare metal electrode swathed in cotton soaked in a vivid purple solution of secret chemical composition. Temporary enthusiasm for the coagulating and deturgescent effects on the nasal mucosa ran high with claims of cures in 70 per cent of the cases. I personally narrowly escaped electrocuting one patient, and so promptly consigned my machine to outer darkness. This novelty rapidly fell into disfavor, a classic example of promising seed falling on stony ground.

Of the 60 papers on nasal allergy 37 have appeared in the last 20 years. The great strides made in a clearer understanding of the fundamental nature of the allergic process, the value of cytological examinations, of methods of skin testing, of careful desensitization, the recognition of the merits of small dosage therapy—all have been made by members of this Society. The formation within recent years of the American Academy of Ophthalmological and Otolaryngological Allergy is a clear indication of how far we have come since the days when an outstanding rhinologist had no better recommendations for his allergic patient than a morning walk around a city block, and a daily colonic irrigation by means of an apparatus which bore the euphonic designation of the Cascade enema bag.

DEAFNESS, TINNITUS, ETC.

The concern of every otologist since the dawn of this specialty has been some form of chronic deafness, however, designated as catarrhal, adhesive, or progressive. Efforts to meet this stubborn and often discouraging problem fill the early pages of the *Transactions*. Centering about the eustachian tube they consisted for the most part of catheterizations, intratubal dilatations, and intratympanic medication with vapors, sprays, instillations and lubrications; mostly, alas, with rather nebulous results. Above all, during the first ten years chief reliance was placed on some form of tubal bouginage, the merits of which were a ceaseless subject of dissension, and divided the Society almost into two armed camps. In 1900 enthusiasm, particularly on the part of its inventor, for a gold tipped and electrically heated bougie provoked strong condemnation from those who were unable to duplicate its results and who could not conceive of any means whereby in 30 seconds a tubal stricture of 10 years' duration could be resolved. Some 40 years later another otologist, in the course of a radical mastoidectomy, came inadvertently on the broken-off tip of such a gold bougie which had perhaps been exerting some dilating effect over the intervening years.

Increased knowledge of the role of the nasopharynx, greatly enhanced with the development in 1910 of the nasopharyngoscope, led slowly to a changing concept toward certain forms of chronic deafness and the recognition of the futility of local treatment of the middle ear and Eustachian tube. Particularly in its progressive form, now known as otosclerosis, it became more and more evident that the cause was a far more deep seated one for which no such simple measures as intratympanic injections of thyroxin would provide the answer. Thus over the years and after prolonged and intensive anatomical and pathological study, the osteitic changes in the labyrinthine capsule were recognized and surgical [illegible] conceived to attack the problem.

With round window grafts as the forerunner in [illegible], the operation of fenestration has since come to constitute perhaps the greatest single advance in the whole history of otology. What it has accomplished in the past 15 years for those afflicted with progressive deafness, need not be mentioned. That the path to its present state of fulfillment has been a thorny one, beset with opposition from many who could speak with otological authority, is evident from the following remark made in 1940 by an outstanding otological pathologist: "From Coast to Coast marked and unwarranted enthusiasm has been expressed for a new technique of opening the horizontal canal. This will not prevent further deterioration.

of the neural end-organ. Dietary regulation, calcium, phosphorus and vitamins to reverse halisteresis and to promote regeneration of the damaged neural mechanism have produced far greater hearing improvement than any fistula operation thus far reported. The ingenious, time-consuming and difficult technique has nothing to do with the success or failure of the operation. Such complexities as mobilization of the drum membrane and removal of the malleus have no point whatever."

Fortunately today we can look back, with the advantage of hind-sight, and appraise this unfavorable criticism as premature and inaccurate. The future of fenestration would appear to be limited only by the ability of human ingenuity to perfect it.

SEROUS OTITIS.

Considering its present day frequency it is surprising to find in these 50 years only five papers on the subject of serous otitis media. One must conclude that either this condition has become increasingly frequent or that it was neglected, either as presenting no essential therapeutic problem or as being of less importance than those posed by many other otological subjects. The same is true of otitis externa, concerning which there is not a single paper before 1910, and only a total of ten in the 50 years. It was not till the second world war that a really thorough investigation of this commonplace disease initiated an interest which has steadily progressed with each passing year.

ACUTE OTITIS AND MASTOIDITIS.

Care and treatment of acute otitic infections were the almost daily concern of every otologist in the early nineteen hundreds, when they were a common sequel of typhoid and scarlet fever and of upper respiratory infections. Despite their frequency there was no essential change in the fundamental methods of therapy until the advent of the antibiotics. The merits of various post-incisional treatments have been a frequent subject of debate, but we find a total of only 40 papers on acute otitis, 25 appearing in the first ten years of the Transactions. The interest in acute mastoiditis has been much more active. Of the 85 papers more than half of them were written before 1916. Leeches and the ineffectual Weil incision soon gave way to the classic Schwartze operation, which remained standard practice for the ensuing 30 years.

In 1905 attempts to avoid the prolonged healing incident to the open post-aural wound led to momentary enthusiasm for the so-called blood clot operation and transitory success in securing primary closure of the incision. The relationship of gastro-intestinal infections to infant mastoiditis was the subject of numerous presentations in the years between 1925 and 1930, since when it seems mysteriously to have faded from the picture. With the advent of chemotherapy the mortality from acute otitic sepsis fell from 25 to 4 per cent, and the incidence of acute mastoiditis by 1942 had fallen to 50 per cent of that of the first 20 years of the period we are reviewing. Only those of you who can look back to your experience of those years can adequately appreciate the drastic and in many respects unique change which has occurred in a situation formerly so common-place.

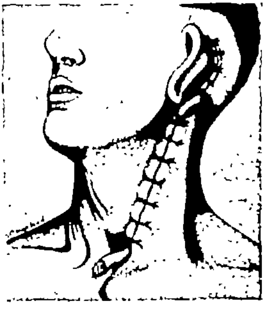

Fig. 1. Cut from an early Transactions showing a common procedure applied to lateral sinus thrombosis (jugular vein resection).

It is, however, in a survey of the complications which so frequently attended acute otitis and mastoiditis that this change becomes so strikingly apparent. Between 1900 and 1930, paper after paper vividly presents the discouraging and often tragic experiences with sinus thrombosis, brain abscess and meningitis. The mortality of such intracranial complications during the early years was appallingly high, despite drastic jugular vein resections, intracranial explorations and all attempts to find some surgical solution to the dread specter of meningitis (Fig. 3). For a masterly dissertation on drainage of the cisterna magna, there was awarded its author as offering a faint hope in this dilemma, one of the only three gold medals ever given to a member.

Accounts of a specialized complication appeared first in 1930, when petrositis became a subject of intense interest with no less than 13 papers considering varied aspects of it during the ensuing five years.

There are in all 115 papers concerning these acute complications of otitic infection. Some idea of the dramatic decline in their incidence can be gained from figures which show that since 1949 to the present date there has been published in the *Transactions* only one paper dealing with such complications. This phenomenal change has presumably been due to the entrance into the therapeutic picture of chemotherapy. Prior to 1930 autogenous vaccines, leucocytic extracts, immune sera, intravenous and intracarotid dyes had all been hopefully tried, but with little consistent effect on the high mortality associated with the systemic and intra-cranial complications which I have outlined.

Not until the advent in 1937 of sulfanilamide did real hope *dawn on the horizon. There followed in rapid succession* prontolyn, sulfathiazole and sulfadiazine, to be succeeded shortly by that drug which perhaps more than any other in medical history, save possibly diphtheria antitoxin, has saved countless lives and revolutionized the whole practice of surgical otolaryngology—penicillin. The many still newer antibiotics which seem to spring up almost overnight may well bring further startling changes in medical practice but penicillin will, I believe, remain a medical and surgical landmark for all time.

CHRONIC OTITIS MEDIA.

The frequent occurrence in the early days of acute otitic infections led automatically to a high incidence of chronic otitis media. In high favor during the first decade of the

Transactions was ossiculectomy, the detailed and meticulous technique of which leaves one profoundly impressed with the manual dexterity and patience of the operator of that period. The separation of stapedial adhesions, delicate disarticulation of ossicular ligaments, and the removal of mucosal granulations—all under the handicap of poor visualization and persistent bleeding—give vivid testimony to the skill possessed by the pioneers of the Society.

In 1901, adopted from the German school, the so-called radical mastoid operation came into vogue, and ossiculectomy, despite good reports in large series of cases, faded slowly from the picture. The technique of the post-aural radical of that day, with its painstaking attention to anatomical detail, and to the removal of every vestige of disease indicate, in my opinion, that the operation of 50 years ago was no whit less well done and yielded fully as good results as any present day procedure. Granted the merits of the later endaural incision, the early radical mastoid operation, with or without skin grafting was a surgical masterpiece, and I am sure that many of you have seen examples of it in patients operated upon during those by-gone years.

It was the frequent post-operative hearing loss which in 1908 led to the vigorous endorsement by many otologists of the modified radical operation, concerning which there was lively debate and frequent disagreement. By some it was declared to be no better than an extremely extensive simple mastoidectomy, by others to be inadequate for removal of middle ear pathology.

The advent in 1942 of the endaural incision and of the electric burr, the latter in frequent use before 1900 and then abandoned as a "relic of barbarism," have led to steady advances in temporal bone surgery, but this survey still leaves me with a feeling of admiration for much of the work of 50 years ago.

The intracranial complications of chronic otitic suppuration follow a close parallel with those of acute otitis. Fatality after fatality is reported after brain abscesses and meningitis, despite the application of every known measure then available. More than half the papers on this subject appeared before 1916, but discouraging results continued until the advent of chemotherapy.

LABYRINTH.

The labyrinth has been the subject of some 60 Triological papers, the bulk of them dealing with labyrinthine physiology and its laboratory investigation. As a surgical complication it occasioned in 1907 a detailed description of labyrinthectomy which, as a model of meticulous and artistic surgery, is the equal of anything to be found in the entire annals of the Society. Over the years there has accumulated a vast amount of knowledge of labyrinthine histology and pathology, and through the enthusiasm and curiosity of certain members of the Society experimental physiology of the labyrinth has led to a greatly increased knowledge of vestibular function and its disorders.

AUDITORY EDUCATION.

For 15 years Triological interest in deafness was concentrated on medical and surgical treatment. It became increasingly apparent that this approach could be of little help to large numbers of old and chronically deafened patients, as well as to thousands of children whose unsatisfactory progress in school remained a problem and whose often serious deafness continued to escape detection. In 1910 began widespread interest by the otologist in the social and educational aspects of this problem, and the development of various methods for helping these handicapped and auditorily crippled individuals. The first crude hearing aid appearing in 1917, and tremendous impetus was given this vast project by the development of the pure tone audiometer between 1921 and 1930.

The tuning fork devotees at first gave grudging ground to this testing innovation, but with its obvious advantages in multiple examination of school children the audiometer won a firm place in the equipment of most otologists. In 1926, largely through the efforts of members of this Society, the American Federation for the Hard-of-Hearing was formed, and since that time gratifying progress has been made in solving many audiological problems, and in rehabilitating thousands of otherwise seriously handicapped individuals. Assistance from audio-physicists and from scientific laboratories has been of immeasurable help to the otologist in this work. In recognition of outstanding service in the education of the deaf child and in the founding of a national institute for this purpose, the Society in 1933 awarded a second of the only three gold medals bestowed since its foundation.

The second World War, with its incidence of auditory damage from gun fire and explosive concussion, furnished still further impetus to rehabilitating men who had been thus severely deafened, and was the initial factor in the evolution of a more specialized branch of otology now known as Audiology. Viewed in retrospect the phenomenal progress made in all aspects of these accomplishments has in large measure been due to the membership of this Society.

TUBERCULOSIS.

Modern methods of public health and education, mass X-ray examination of the chest and availability of certain antibiotics have done much in the past ten years to reduce the incidence of pulmonary tuberculosis, and as a corollary, of laryngeal tuberculosis. During the first two decades of the *Transactions* no such encouraging situation existed. Of the 63 papers on some form of otolaryngological and chiefly laryngeal tuberculosis, five-sixths were presented during the first 30 years of the half century, and only five during the last 15 years. The laryngologist of 1896 was quite accustomed to the use of the stethoscope, an instrument notably missing in the instrumental exhibit of the last meeting of the American Academy. Since the early days the treatment of laryngeal tuberculosis has covered a wide range of procedures.

Added to systemic prescription of sea air and a felicitous climate have been such varied local measures as topical lactic acid, surgical curettage, tracheotomy, laryngeal nerve injections, actual cautery, diathermy and instillations of chalmoogra oil. All these achieved but indifferent success, and it was not until 1925 that the introduction of artificial pneumothorax, primarily applicable to pulmonary tuberculosis, began to effect a striking reduction in the incidence of its laryngeal complications. The perennial argument as to the existence of true laryngeal tuberculosis appears to have been answered in the negative. The beneficial results of pneumothorax are clearly evident in a report in 1930, in which a series of 5,400 patients with pulmonary disease developed laryngeal complications in only 12 instances.

Much the same situation obtains in the case of syphilis. Involvement in this disease of the tonsil, the turbinates, the larynx, the tracheobronchial tree and the auditory nerve had been made the subject of fairly regular reports up to 1933. Since then the otolaryngological manifestations of syphilis have with constant improvements in antisyphilitic therapy markedly decreased, and there have been no papers on the subject during the past ten years of the half century.

SPEECH.

Interest in speech defects, aside from those stemming from laryngeal pathology, is today largely centered in educational institutions. The first 20 years of the *Transactions* contain frequent articles on such vocal disturbances as stuttering, aphasia and speech defects of psychogenic origin. Indeed, it was for the establishment of a special institution for the rehabilitation of patients with a wide range of such speech defects that the Society in 1940 awarded the third of the three gold medals which have thus far recognized an outstanding achievement of one of its members.

It was not till 1943 that interest in training laryngectomized patients to produce a satisfactory esophageal voice reached its present wide application. Speech training for the profoundly deafened patient has today come to fall almost entirely within the newly developed field of audiology, to which I have already referred.

BIOCHEMISTRY.

A study of Triological papers up to 1920 shows a predominant interest in the surgical aspects of otolaryngology, in operative technique and in the results of such surgical approach to diseases of the nose, sinuses, throat and ear. The surgeon, so to speak, had the floor. It was, therefore, with a profound shock that this Society in 1922 heard a report, based on replies to a questionnaire, detailing no less than 332 non-anesthetic deaths incident only to intranasal surgery and to tonsillectomy and adenoidectomy. Of these deaths 120 were due to meningitis, 56 to hemorrhage, 56 to sepsis, and 43 to embolism. Scarcely had the repercussions of this grim news subsided when a supplementary report the next year added 112 additional deaths from similar operations.

Papers of the ensuing years show a pronounced trend toward a more conservative attitude, particularly in reference to nasal surgery, as exemplified by the following comment: "Through rhinological enthusiasm the nose has been looked upon as the probable source of every human ill, and the ruthless slaughter of intranasal structures, regardless of physiological function, has been universally practiced. Happily a saner view is prevailing and the belief is growing that epilepsy, asthma, dysmenorrhea and arthritis may have other causes than a point of pressure within the nasal fossa."

Beginning in 1932 there began a growing feeling that far more attention should be paid to the medical rather than to the surgical side of the otolaryngological shield. For years before this, sporadic attention had been called to the relationship between pathology of the ear, nose and throat and the gastrointestinal tract, to a so-called lithemic diathesis and to the importance of a patient's mode of life, his physical activity and his food selection.

Autointoxication was considered to have an intimate relationship to the growth of lymphoid tissue and to the physiology of the nasal mucous membrane. These straws in the wind had begun, about 1925, to accumulate into what came to be known as the biochemical approach to otolaryngological problems.

More and more was written about vitamins, acid and ash base balance, the autonomic level, sympathetic and parasympathetic dominance, the suprarenal, thyroid and pituitary glands and the profound effect of systemic physiology on disorders of the ear, nose and throat. Therapeutic innovations of radical changes in diet, of minute doses of insulin, hydrochloric acid, and iodine were reported as highly successful, not alone by isolated enthusiasts, but by nation-wide groups with irrefutable testimony to the efficacy of these novel measures.

To be sure the biochemists and physiologists themselves looked rather askance at what seemed to them to be assumptions without scientific proof, but clinically the medical approach seemed for the moment to be outstripping the surgical as the order of the day. The past ten years have shown a marked abatement in this enthusiasm for biochemistry, but unquestionably it has exerted a salutory curb on unnecessary surgery. Meanwhile the concept that a large number of otolaryngological ills have a psychosomatic basis is striving for its place in the center of the stage.

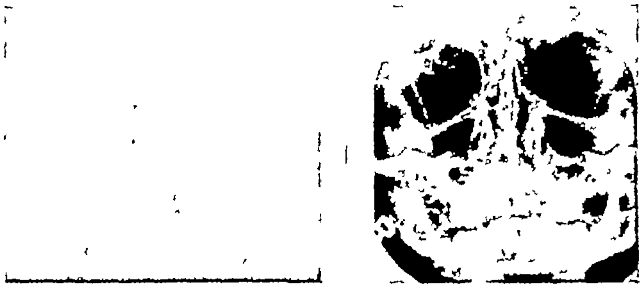

Fig 4 A comparison between a nasal sinus x-ray of 1906 and one of 1953

PLASTIC SURGERY.

An interest in plastic and reconstructive surgery, especially of the nose, was well established in the *Transactions* before 1901. Repair of cleft palates was a hobby of a few members, although this field has tended more and more to fall within the orbit of general or pediatric surgery. Chiefly applicable at first to the correction of cosmetic defects but now applied to a betterment of nasal physiology, modern rhinoplasty has so expanded as a subspecialty that there is today an American Society of Oto-Rhinological Plastic Surgery to serve the needs of those who make this field a major part of their work.

X-RAY.

The value of the diagnostic information afforded the modern otolaryngologist by X-rays of the sinuses, mastoids, larynx and esophagus is taken largely for granted. This situation has come about only with the steady perfection of the technical apparatus and increasing perfection in skillful interpretation by the roentgenologist. In 1897, films of the larynx were regarded as a waste of time, those of the nasal sinuses as of no value beyond determination of the size of the cavities. A comparison between a sinus film of 1906, and one of 1958, will serve to explain the clinician's early lack of enthusiasm for roentgenology (see Fig. 4).

Gradually skepticism as to the reliability of the X-ray and its usefulness in affording dependable information as to existing pathology gave way to progressive acceptance, so that by 1931, it was listed as among the most important of all diagnostic aids.

For the therapeutic application of the X-ray, *Transaction* papers show far less enthusiasm. X-ray treatment of tonsil pathology, of acute sinusitis and even of lymphoid tissue hypertrophy, though sporadically successful, must still be considered of dubious value.

ANESTHESIA.

The modern otolaryngologist has but little concept of the problems incident to anesthesia during the early years of the Society's existence. Chloroform was largely the anesthetic of choice, but entailed an alarming mortality so that patients stood in terror of it and often elected to forego any anesthesia. Cocaine was available as a substitute and was used not only locally, but also by injection, even in children in what today would be regarded as dangerous concentrations. It is understandable that one physician gave up such local anesthesia because "of the screaming of the patient, the fright of the parents, and the general excitement." Ether, by 1910, had by virtue of its far greater safety, largely superseded chloroform, and novocaine had replaced cocaine in local anesthesia except for surface application. From 1930 on, the whole specialty of anesthesia made tremendous strides, and intratracheal and intravenous modifications made possible serious surgical procedures which would otherwise have been either impossible or extremely hazardous.

ANATOMY AND RESEARCH.

During the first decade of this Society's existence the bulk of the papers dealt with its members' clinical experiences. These men were almost exclusively busy practitioners, working in hospital clinics and in their private offices. Interesting case reports of unusual clinical entities, of therapeutic successes and failures, of operative techniques and results, formed the majority of the papers presented during this period. Within the past 20 years there has been a pronounced trend toward presentations of anatomical, histological and pathological investigations and research, often of a most detailed and time-consuming nature, and covering such a wide range of subjects and laboratory experiments as to be far beyond the time available to cover them.

Some few of these have come from men primarily in academic positions, teachers in medical schools, or research workers attached to large hospitals. It is stimulating to note, however, that the majority of such investigative and research work has been done by men engaged in the every day practice of medicine with its accompanying demand on their time and its interruption of their day's routine. While his lay friends and businesss associates were happily seated at the bridge table, many a Triological worker had burned the midnight oil in search of microscopic signs of atrophy in a guinea pig's tectorial membrane, or in observing the ciliary activity of a rabbit's nasal mucosa, fired with a zeal for adding even one new observation or ray of light to the ever expanding horizon of the specialty.

Much of this work has been done by men who have never occupied the otolaryngological limelight, but whose interest, curiosity and enthusiasm have led to often outstanding contributions to medical science. Of these papers covering a wide range of non-clinical subjects, two-thirds have appeared during the last 20 of this 50-year period, half of them between 1926 and 1936, a period when broader fields of bacteriology, experimental physiology, advanced photography and the ultramiscroscope were contributing enormous help to scientific investigators.

INSTRUMENTS.

Scattered repeatedly through the *Transactions* are cuts and descriptions of a wide range of surgical instruments, many of commendable ingenuity and all designed to meet some hitherto unsolved surgical problem. Such is the individuality of medical practice that a tool enthusiastically championed by one operator is viewed with skepticism or even scorn by a confrere. Surveying the large assortment of these instruments, I fear that the majority of them have found lasting favor only with their inventor, and having made a brief appearance in some catalogue, have since disappeared and so been forgotten by posterity. Were I asked to name any single product of an ingenious and imaginative mind amongst all those figured in the volumes of the past half century I can think of none which to my mind surpasses the original model of the Mosher safety-pin closer.

UNSOLVED PROBLEMS.

In the 1924 volume there appears a challenging list of what were termed unsolved problems of otolaryngology. It included chronic deafness, meningitis, the perfection of hearing aids, the common cold, malignant disease of the nose, throat and esophagus, atrophic rhinitis and the X-ray treatment of hypertrophied lymphoid tissue.

This list was subjected to review with, happily, an elimination of a few items and with clear evidence of progress toward the solutions of several others. The common cold remains as much of an enigma as ever, and tinnitus and a completely satisfactory solution of the riddle of frontal sinus surgery still deserve a high rank in the list first drawn up 30 years ago.

DISCUSSIONS.

The number of pages given over to the discussion of the papers has fluctuated but little. In the early days this tended to be more free and informal, with many more members taking part, (in 1902, with 243 members, there were 234 discussions), rising spontaneously, indeed irresistibly, often to disagree vigorously with the pronouncements of the essayist. Papers were often, willy-nilly, converted into a panel discussion with questions and answers flying thick and fast, sometimes with more heat than light. Such verbal interchanges at times occupied more *Transactions* space than the very paper itself.

Today these impromptu debates and contentious polemics have been largely replaced by formally prepared discussions by one or two selected discussers who tend to acquiesce with the author's point of view, and so deprive the meeting of those occasional pyrotechnic displays of honest but frank dissension which amused the audience if not the essayist.

A former president once charged a former editor with deletion from the *Transactions* of all those sparkling bits of repartee with which certain members, and incidentally himself most of all, were wont to color the meetings. I am sure that your present editor will only too gladly include such occasional levity in our otherwise serious deliberations, but of late there has been a lamentable paucity of such salutary humor.

There was much, I believe, to be said for the oldtime informal discussion. Today with the inevitable secretarial scrutiny of the clock, the inclination for the general membership to take part in discussion is chilled and so abandoned. Younger men, often with something valuable to contribute, hesitate to rush to the rostrum, fearing either lack of importance of their remarks or of consuming precious time needed for completion of the program. Added to this is the fact that, with increasing specialization within the specialty, a paper on the complexities of bone conduction does not stir a thought in the mind of a man whose primary interest is cancer of the larynx. In 1900, any otolaryngological subject was the lively concern of all the members and was provocative of personal contributions by a large proportion of the audience.

THE EDITOR.

The mechanics of publication of the *Transactions*, for years a perennial problem, are now functioning with heretofore unattained smoothness under the masterful hand of your present editor who, through utilization of the facilities of THE LARYNGOSCOPE, is able year after year to present the completed volume almost before the discussers of the papers have ceased to doubt that they actually said what the stenographer took down.

You will doubtless note that I have in this survey of so many outstanding contributions to otolaryngology made by past members of this Society, refrained from the mention of individual names. Granted that some stars have shone with greater brilliance in the otolaryngological firmament than others, nevertheless, so many workers long since forgotten have done so much to illumine the darkness that it would do them grave injustice to single out only planets of the first magnitude. These latter are known to all of us and need no special mention to assure them of their permanent place in Triological history. Fortunately, there are today a few of these pioneers who are living to enjoy the fruits of their labors and to witness the realization of their hopes and fulfillment of their dreams.

It is a saddening thought, however, to recall that of the 23 past presidents of the Society, so felicitously saluted in 1928 by the only poem to be found within the covers of the *Transactions,* only two are still living.

GENERAL IMPRESSIONS.

One could scarcely complete such a journey through the literary past without being left with certain impressions and emotions. Of these my strongest feeling is one of admiration for, and appreciation of, the courage and fortitude with which the earlier workers in this field met the difficulties, the defeats, the obstacles and disappointments which beset them on every hand in days when our modern facilities and technical accessories were unavailable.

I have admired the detail and meticulousness with which many men applied themselves to the study of a subject which might, on superficial consideration, seem to offer little material on which to work. One paper on the normal tympanic membrane covered over 40 pages.

I have been impressed with the conscientiousness and dogged persistence of the man who admonished any operator doing an ossiculectomy to search for and remove every last vestige of a necrotic incus or shred of diseased mucosa, no matter if it took all day.

That some of the pioneer laryngologists were, like Kipling's shipwrecked mariner, men of "infinite wisdom and sagacity" and likewise of resourcefulness, is attested by the story of the physician who, unexpectedly found himself faced with the necessity of doing an emergency tracheotomy in a barnyard. Lacking any substitute for a tracheal tube, he is said to have seized a passing goose, extracted from its wing a large feather, and cut off and sharpened the quill with his pen-knife. Having opened the trachea he inserted this heavensent make-shift cannula, all within the space of less than a minute.

I have been further impressed with the wide range of interests and medical hobbies in what at times has been criticized as a narrow specialty. These interests have run the gamut from thyroid surgery to audiophysics, from cleft palates to esophageal resections. They range from studies in experimental bacteriology to social and educational projects of far-reaching benefit to humanity at large. They include excursions into such allied fields as allergy, biochemistry, neurosurgery and dietetics.

Perhaps above all else, I have been impressed with the boundless enthusiasm which through the years has impelled men who, though contributing fully in their daily work, have yet sought to go the second mile toward the scientific advancement of the specialty.

CONCLUSIONS.

With the enormous changes which have taken place during the past 50 years, which I have sought, even in so fragmentary a way, to encompass, it is an intriguing though perhaps unwise temptation to speculate as to the progress and innovations which will come to pass during the next half century of Triological history. A fortunate few of you may, with the promised help from geriatrics, be able, as I have done, to review a similar period. If so, you will no doubt have seen even more astounding changes incident to newer antibiotics, to the medical uses of atomic energy, to the triumph of radiation over malignant disease and to a yet unknown array of measures directed to the betterment of human health. You may, though the prospect seems dim, be able to look back on the conquest of the common cold.

Far be it from me to elect the role of a prophet who today, as in the days of old, stands in such danger of being stoned from the market place. I have thought it of value. and I hope of some interest, to take stock of our past accomplishments lest we become either too complacent or arrogantly satisfied with what today, as well as at every past moment in our professional history, has been called "modern medicine."

Whatever miraculous discoveries may lie ahead, the otolaryngologist will, I am sure, do well to continue to place himself beneath the sheltering wings of the three guardian angels of the doctor—Common Sense, Surgical Judgment, and the Study of the Individual.

If in this brief time I have left with you a necessarily cursory and somewhat superficial view of the achievements of the members of the Society over the past half century, I can assure you that the attempt thus to review the *Transactions* has for me been a liberal, rewarding, and I fear, long overdue experience in post-graduate education.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

A joint meeting of the North Carolina Eye, Ear, Nose, and Throat Society and the South Carolina Society of Ophthalmology and Otolaryngology will be held at Durham, N. C., November 4, 5, and 6, 1954. Headquarters will be the Washington Duke Hotel.

Guest otolaryngologists will be Dr. Stanton A. Friedberg of Chicago, Ill., Dr. John Maxwell of Ann Arbor, Michigan, Dr. George Bayling of Durham, N. C. The following guest ophthalmologists will likewise be on the program: Dr. Charles Iliff of Baltimore, Maryland, Dr. John McLean of New York, N. Y., and Dr. Townley Paton of New York, N. Y.

On Wednesday preceding the beginning of the meeting, operative clinics will be held in the various hospitals of Durham and Chapel Hill.

Finally, there will be a football game on Saturday afternoon between the University of North Carolina and the University of South Carolina in Chapel Hill.

SECONDARY SKIN GRAFTING IN FENESTRATION AND MASTOID CAVITIES.*†

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and
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(By Invitation).
Houston, Texas.

The development of fenestration surgery has progressed through several phases. In the first or pre-surgical phase, much discussion and study occurred concerning the possibility of establishing a definite diagnosis of otosclerosis as a cause of conductive deafness; in the second phase the emphasis was on the actual technique of the operation; a third phase was again investigative, concerning the more accurate diagnosis of the condition, and better selection of satisfactory candidates for operation, comparison of pathology found during the operation with preoperative tests, and prognostic details concerning anticipated results; a fourth phase concerns emphasis on techniques to promote rapid and complete healing of the fenestration cavity.

An early method employed to promote rapid healing of the cavities was strict asepsis during surgery and postoperative treatment. This gave improved results, but left something to be desired, as the complete healing of the cavity with a lining of squamous epithelium was often a long and arduous course. Some failures for complete healing with a true skin lining were experienced. Most of these cavities finally became dry; but many were prone to recurrent drainage, due to areas in the lining membrane which would macerate quickly whenever contamination of the cavity occurred, and these areas often responded to treatment slowly. Juers[1] suggested that these areas were not completely covered with true squamous epithelium, but may have been covered with

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metaplastic mucous membrane which originated from the mastoid cells and covered the surface before true skin growing in from the meatus and canal covered the cavity.

At any rate, cavities healing in this fashion would often show poor resistance to infection, which was an ever-present danger before complete healing occurred. These infected cavities, in spite of asepsis in cleaning postoperatively, markedly lengthened the healing time. A frequent additional discouraging factor was the repeated visits for treatment, often requiring travel for long distances because for various reasons, aid was not accessible in the local area. It is superfluous to state that a draining ear is disillusioning to the patient, regardless of the improvement in hearing obtained, and is not an inviting prospect for the fenestration candidate.

Primary skin grafting of the fenestration cavity to promote healing has recently been used by some operators, but according to our previous experience and the experience of others[2,3] in primary grafting of mastoid cavities, incomplete healing often occurred, resulting in cavities similar to the ones described as healed without grafting. We also emphasize that a cavity which appears to be healed and dry soon after primary grafting does not always stand the test of time and trauma from contamination, without revealing areas of breakdown in the membrane with resultant drainage. An additional factor against our using this method is our belief that as soon as the fenestra is made, the flap should be immediately and tightly packed in place if postoperative labyrinthitis is to be kept at a minimum. For these reasons we were reluctant to use this method.

For the purpose of this paper a cavity is not described as healed unless complete epithelialization of the cavity with true, healthy squamous epithelium is present. Padgett[4] has stated, "on cancellous bone, only 50 per cent of a skin graft will take, and as much of a thick graft will take as a thin graft," and emphasized that "if the blood supply of the base is deficient some formation of granulations, providing they are kept clean, may enhance the certainty of a 'take'."

Padgett's description of the healing of a skin graft on a granular surface illustrates the reason for the rapid and complete healing of the secondary graft in a mastoid or fenestration cavity, and is as follows:

"The graft is first fixed in place by an exudate of fibrin which results in adherence to the base within five hours, and plasmic circulation in 24 to 48 hours, which temporarily preserves the viability of the graft. Soon the endothelium of the blood vessels of the host area sends proliferating buds toward and into the graft, some endothelial spaces in the graft connecting up with the endothelial buds.

"The stages of vascularization begins at about 18 hours and from then on is progressive; within 65 to 70 hours definite continuity of the blood vessels of the host and those of the central part of the graft can be noted. By the eighth day the blood supply is nearly complete, and by the tenth day the fibroblastic connection is fairly full and complete.

"Some exfoliation is likely to occur; the most superficial layers of the epithelium tend to show some degenerative changes. The degree of exfoliation is in direct proportion to the speed of the revascularization of the graft. The granular surface should be clean, firm, red in color, and not too exuberant and need not have a bacterial count which is absolutely negative. When the wound is clean, the bacterial count of the secretion will not be high."

Our experience with the "taking" of secondary grafts in fenestration and mastoid cavities has closely followed Padgett's description of grafts healing on granular surfaces. When applied with good technique, under optimum conditions, the healing has been rapid and complete with very little exfoliation, the skin appearing clean, soft and pink seven or eight days after the graft was placed. While the idea of secondary grafts[5,6] in mastoid cavities is not new, our method of application differs from those previously described.

To our knowledge secondary grafting in fenestration cavities has not been previously described. Previous descriptions of secondary grafting applied to mastoid cavities entailed cumbersome procedures requiring a general anesthetic. In our technique a split-thickness graft is obtained from a donor site on the under surface of the upper arm where the skin is thin, pliable and contains very few hair follicles. The anesthesia required for the donor site is local injection of approximately 10 ccs. of 1 per cent novocaine. The type of graft taken is a split-thickness graft which is mounted on Owens Surgical Fabric as suggested by Farrior[7] in primary grafting. Owens Surgical Fabric may be obtained from Winchester Mills, Inc., 120 West 42nd Street, New York 26, N. Y.

The skin is held in place on the fabric by the use of an ointment such as aureomycin ointment, to prevent the graft from curling. These grafts are applied through the endaural meatus in six or eight strips of appropriate length and width, first covering the tegmen, then the posterior wall of the cavity, the anterior wall of the external auditory canal, the facial ridge, and lastly the soft tissue of the lateral wall of the cavity and the bare areas in the meatus. Careful attention is directed to intimate contact of the graft with the granul area of the mastoid cavity.

The cavity is usually ready for grafting anywhere from the tenth to the eighteenth day postoperatively, as a [illegible] firm layer of granulation tissue usually lines the cavity by this time. The wet surface of the granulation tissue opposed to the wet raw surface of the graft allows the graft to adhere snugly to the base by capillary attraction. The graft [illegible] moved along this surface of the base to the appropria[illegible] tion without difficulty. Attention is called to the [illegible] packing of the cavity for fixation of the graft is [illegible] sary in this method. The fabric is removed [illegible] seven to fourteen days after grafting, by [illegible] the graft or by lifting up the edge of the [illegible] ear hook and carefully removing the fabric.

Results of grafting in fenestration and mastoid cavities, all of which have been grafted for more than a period of six months, and some for a period of 20 months:

Fenestrations		60
Completely healed in two months or less	48	
(Of these, 37 healed in four weeks or less)		
Healed in three months	1	
Healed in four months	1	
Healed in five months	3	
Healed in six months or over	4	
Unhealed after 12 months	3	
Modified Radical Mastoidectomies		14
Healed in two weeks	8	
Healed in four weeks	3	
Healed in four months	2	
Healed in seven months	1	
Radical Mastoidectomies		6
Healed in two weeks	2	
Healed in two months	1	
Partially healed except middle ear	3	
Fenestration cavities not grafted		100 cases
Average healing time, 4 4 months.		
Secondary skin grafts		60 cases
Average healing time, 1 7 months		

SUMMARY.

In summary, secondary skin grafting is a relatively simple office procedure which results in a reduced healing time for fenestration and modified radical mastoid cavities in the large majority of cases. The advantages of the method described are: ease of obtaining skin from the arm, the area is not exposed and is not a cosmetic problem; the donor area involved does not appreciably handicap bathing postoperatively; a "rapid take" with complete healing by the epidermal layer of squamous epithelium, which can withstand contamination without breakdown of the cavity lining is possible, and packing of the cavity is avoided. The method may also be employed to cover an area of an unhealed cavity which presents a true granulating surface, several weeks to months following surgery. In the radical mastoid cavity much promise exists for the use of the secondary graft to promote rapid healing of the cavity, due to the fact that specific agents can be used locally to rid the cavity of infection before the graft is applied.

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AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1 and continue for a period of ten months. *Registrations* must be completed before August 15. Detailed information and application forms may be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minnesota.

The Department of Otolaryngology, University of Illinois College of Medicine, announces its Annual Assembly in Otolaryngology from September 5 to 11, 1954. The entire week will be devoted to surgical anatomy and cadaver dissection of the head and neck, and histopathology of the ear, nose and throat. The Assembly will be under the direction of [illegible] F. Snitman, M.D.

Registration will be limited. For information [illegible] Department of Otolaryngology, University of Illinois [illegible] of Medicine, [illegible] Street, Chicago [illegible]

CLINICAL TRIAL OF AN ANTIBIOTIC-DECONGESTANT-ANTIHISTAMINIC SOLUTION FOR THE TOPICAL TREATMENT OF POLLENOSIS.

MARTYN A. VICKERS, M.D.,
Bangor, Maine.

An increasing amount of evidence incriminates an infectious component as a significant factor in the problem of the satisfactory treatment of so-called allergic rhinitis. In many instances, particularly in the perennial variety of allergic disease of the nose, and also in seasonal allergies or pollenosis, the infectious component appears to represent secondary bacterial invasion of tissues whose defense mechanisms have already been weakened by their intense allergic inflammatory reaction.[1] It is also conceivable that pre-existent infection may render the nasal mucosa more reactive to exposure to allergic incitants.[1]

The association between allergy and infection may be more frequent than is usually recognized. Although from the patient's history, demonstration of reaction to exposure to allergens and appearance of the nasal mucosa, any competent diagnostician will readily determine the existence of allergy and also the presence of patent infection, he may overlook latent and apparently asymptomatic infection without resort to precise laboratory procedures. Thus a number of investigators have commented on the fact that such cultural studies have not infrequently disclosed the presence of unquestioned nasal pathogens in patients whose histories, clinical examinations and other laboratory tests had led to the diagnosis of an uncomplicated allergy.

This not infrequent co-existence of allergy and infection poses definite therapeutic implications. Obviously, from the viewpoint of the overall welfare of the patient, treatment must be directed not only toward the control of allergic symptoms, but also to the eradication of the infection as well. In view

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of a growing belief that such infection, inapparent as it may be, aggravates or prolongs the allergic state, satisfactory control of the latter becomes impossible without simultaneous treatment of the infection. This fact has recently been *brought out very dramatically in a study of patients with allergic* rhinitis in whom the infectious component escaped detection by the usual methods of examination and was revealed only by precise cultural procedures. In these patients, simultaneous short-term anti-infectious and anti-allergic treatment resulted in permanent remission from their symptoms of allergic rhinitis even after all treatment was stopped. The investigators attributed this unexpected finding to the thesis that in some manner the unrecognized infection had rendered the patients susceptible to the allergic reaction and that eradication of the infection was accordingly indirectly responsible for the disappearance of the allergic symptoms.[1]

Most physicians are content to establish a diagnosis of allergic *rhinitis and treat the patient for this condition alone;* however, since an infectious component may be a contributory factor, it becomes exceedingly important to have available a medication ideally adapted for the safe and effective treatment of either allergic or infectious rhinitis or combinations of both. Its use in the presence of secondary bacterial invasion would insure maximum opportunity to combat the nasal condition, regardless of its nature.

Recently, a number of reports have stated that Biomydrin Nasal Spray* provides eminently satisfactory results as an agent for the topical treatment of either allergic or infectious rhinosinusitis.[2,3,4] The efficacy of this antibiotic, anti-histaminic, decongestant mixture* is largely due to the presence of Thonzonium bromide, a mucolytic, wetting agent which is claimed to enable the other constituents to penetrate through nasal secretions that might otherwise prevent its intimate contact with the nasal mucosa itself. By virtue of its wetting action, it is also said to promote the spread of the drug over the entire affected area, penetrating into each crevice and fold and to cling in prolonged contact with the diseased tissues. From the view of the allergist, it has the additional virtue that its antibiotics—neomycin and gramicidin—not only have an extremely low index of sensitization but also, since they are not used systemically, even should sensitization be induced, their topical use would not deprive the patient of an agent that might later be needed for the treatment of a serious, systemic disease.

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* Biomydrin Nasal Spray, kindly supplied by the Nepera Chemical Co., Inc., Yonkers 2, N. Y., has the following formula: Thonzonium bromide—0.05%, Neomycin sulfate—0.1%, Gramicidin—0.005%, Phenylephrine HCl—0.25%, Thonzylamine HCl—1.0%.

Although excellent results have been reported on the use of Biomydrin in allergic rhinitis, the most extensive studies have been concerned with its use in bacterial infections of the nose and sinuses. In view of the foregoing discussion, we believed it of interest to evaluate Biomydrin in a series of patients with seasonal allergic rhinitis (pollenosis) who otherwise would have been treated according to the conventional methods utilized in this clinic and described elsewhere.

METHODS.

The effectiveness of Biomydrin was evaluated during the late summer season of 1953 in a selected series of 75 patients with pollenosis. All of these patients were "new" ones with no previous desensitization treatment histories, and they were further selected in that all were patients whose nasal symptoms had not previously been controlled through oral antihistaminic therapy alone. The nature of their sensitivities was determined by conventional methods, and 39 were found due to grasses, 36 to ragweed, but during treatment and evaluation no further distinction was drawn between the two.

During the study all other medication, including antihistamines, was withdrawn, and Biomydrin was the sole agent prescribed for their previously unrelieved rhinitis. Biomydrin packaged in a plastic spray atomizer was found to be ideal from the standpoint of self-administration, and the patients were instructed to use it as a nasal spray as often as was required for relief of their nasal symptoms. For a single dose, three to four sprays were given in each nostril. The dosage frequency varied from once daily, in one patient, to every three hours in a few cases. The average patient used it four times daily. The span of treatment extended from thirteen to twenty-five days. During treatment, the patients were re-examined at intervals for subjective and objective effects.

RESULTS.

The results of treatment, as indicated by its effect on the nasal symptoms, are indicated in Table I. It can be seen that treatment with Biomydrin was very effective, relieving the symptoms of nasal congestion, sneezing, itching of the nose and rhinorrhea in 60 or 80 per cent of all patients. Of these, 38 or 51 per cent enjoyed complete relief from their complaints. Twenty-two, or 29 per cent testified to adequate relief and were maintained in a state of satisfactory comfort. Twelve, or 16 per cent, stated that their complaints were not satisfactorily controlled. In three, or 4 per cent, the symptoms were aggravated during therapy.

TABLE I

THE EFFECT OF BIOMYDRIN IN POLLENOSIS

	Total Patients Treated	Excellent to Complete Relief	Adequate Relief	Not Improved	Worse
Number	75	38	22	12	3
%	100	50.7	29.3	16.0	4.0

The above data refer to the effect of Biomydrin on the nasal congestion, sneezing and running and itching nose.

Objective examination confirmed the subjective reactions of the patients. Where relief had been obtained, the [illegible] and pallor of the nasal mucosa had disappeared with a resultant relative dryness.

Reactions were relatively infrequent and of a [illegible]. Ten patients complained of varying degrees of [illegible] five of these found the irritation insufficient to warrant discontinuation of treatment as they felt the benefits [illegible] weighed any ill effects. The same attitude was [illegible] of the twelve patients who reported some degree of [illegible] dryness but who continued to use the drug for its [illegible] effects. Four patients complained of some [illegible] ness in the back of the throat after using the drug.

thetic effect of thonzylamine hydrochloride and its presence in the throat can only be attributed to too vigorous a spray. Excessive drying undoubtedly results from the too frequent application of the drug. It is often difficult to asses "irritation" since many patients will complain of this after use of a normal saline spray.

These complaints emphasize the fact that any preparation for topical application to the nose, if used intemperately and indiscriminately, will inevitably produce some untoward effects. It is, therefore, important that patients be instructed in the proper usage of Biomydrin and that they should not be permitted to use it for indefinite periods of time.

SUMMARY AND CONCLUSION.

1. Seventy-five patients suffering from pollenosis due to grass and ragweed, whose symptoms had previously not responded to oral antihistamines, were treated with Biomydrin Nasal Spray.

2. Eighty per cent of these patients experienced adequate or complete relief from their nasal symptoms.

3. Such undesirable reactions as were encountered are believed to be due to the intemperate and excessive administration of the drug.

4. Biomydrin Nasal Spray seems to have definite value as an agent for relief of pollenosis or pollenosis-like syndrome when used properly and not in excessive amounts.

5. Because of its ability to relieve pollenosis and, at the same time, to control secondary bacterial invasion, Biomydrin also seems to be a valuable drug for the treatment of perennial nasal rhinitis, which so frequently is complicated by secondary bacterial infection.

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4. LAZAR, A. M., and GOLDIN, M.: *E.E.N.T. Monthly*, 32:512-515, 1953.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

July 1, 1954.

Acousticon Models A-17, A-180 and A-185.
Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 1, New York.

Auditone Models 11 and 15.
Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67 and 70.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac Model M; Mono-Pac Model "Lyric"; Mono-Pac Model "Rhapsody."
Manufacturer: Beltone Hearing Aid Co., 2900 West 36th St., Chicago 32, Ill.

Cleartone Model 700.
Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Junior Model D-2; Dahlberg Model D-3 Tru-Sonic; Dahlberg Model D-4 Tru-Sonic.
Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Fortiphone Models 19-LR; 20A; 21-C and 22.
Manufacturer: Fortiphone Limited, Fortiphone House, 247 Regent St., London W. 1, England.
Distributor: Anton Heilman, 75 Madison Ave., New York 16, N. Y.

Gem Hearing Aid Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.
Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.
Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico Model J; Maico Top Secret Model L; Maico Maxitone.
Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis, Minn.

Micronic Model 303; Micronic Model "Mercury."
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Model T-10; Microtone Model T-612.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

Normatone Model C and Model D-53.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.

Distributor: Normatone Hearing Aid Co., 22 East 7th St., St. Paul (1), Minn.

Otarion Models B-15 and B-30; Otarion Models F-1, and F-3; Otarion Model H-1; Custom "5."

Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40. Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny Myte).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Model 62 Starlet; Model 72; Model 82 (Zephyr).

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Distributor: Radioear Corp., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Silvertone Model H-16,. J-92; Silvertone Model P-15.

Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.

Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966; Sonotone Model 977; Sonotone Model 988.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Televox Model E.

Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 953; Telex Model 1700.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.
Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster; Model Cameo.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal; Zenith "Regent."
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices have vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TRANSISTOR HEARING AIDS ACCEPTED.

Acousticon Model A300; 1 transistor, 2 tubes. Model A-310; 1 transistor; 2 tubes. Model A-330; 3 transistors. Model A-335; 3 transistors.
Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 35, New York.

Audivox, Model 71; 3 transistors.
Manufacturer: Audivox, Inc., 123 Worcester St., Boston 18, Mass.

Beltone Concerto Model; 3 transistors.
Manufacturer: Beltone Hearing Aid Co., 2900 W. 36th St., Chicago 32, Illinois.

Maico Transist-Ear, Model O; 3 transistors.
Manufacturer: The Maico Company, Inc., 21 N. 3rd St., Minneapolis 1, Minnesota.

Micronic "All American" Hearing Aid; 3 transistors.
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Model T1 (Red Dot); 3 transistors. Microtone Model T1 (Yellow Dot); 3 transistors. Microtone Model T31 (Micro-Mite); 1 transistor and 2 tubes.
Manufacturer: The Microtone Corporation, Ford Parkway on the Mississippi, St. Paul 1, Minn.

Otarion Model C-15; 1 transistor, 2 tubes. Otarion Model D-1; 3 transistors. Otarion Model F-22; 1 transistor, 2 tubes.
Manufacturer: Otarion, Inc., 4757 N. Ravenswood Ave., Chicago 40, Ill.

Radioear Model 820; 3 transistors.
Manufacturer: E. A. Myers & Sons, Inc., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Silvertone Model H-25; 3 transistors.
Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.
Distributor: Sears, Roebuck & Co., 925 South Homan Ave., Chicago 7, Illinois.

Sonotone Model 1010; 1 transistor, 2 tubes. Model No. 1111; 3 transistors.
Manufacturer: Sonotone Corporation, Elmsford, N. Y.

Telex Model 954; 1 transistor, 2 tubes. Telex Model 956; 3 transistors.
Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Unex Model TR-3D; 3 transistors.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Zenith Model Royal-T; 3 transistors. Zenith Model Super Royal-T; 3 transistors.
Manufacturer: Zenith Radio Corp., 5801 W. Dickens Ave., Chicago 39, Illinois.

SEMI PORTABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).
Manufacturer: A. M. Brooks Co., 1222 W. Washington Blvd., Los Angeles 7, Calif.

Aurex Hearing Aids (three types).
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.
Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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(Secretaries of the various societies are requested to keep this information up to date).

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Meeting: Hollywood Hotel, Hollywood, Fla., March 17-18, 1955.

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MORTALITIES AND MORBIDITIES FOLLOWING 20,000 TONSIL AND ADENOIDECTOMIES.*

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This is a discussion of the mortalities and morbidities following approximately 20,000 tonsil and adenoidectomies performed in a 30-year period by general practitioners and otolaryngologists, in Portland, Maine, a community of some 100,000 persons. Such a discussion is important, because these operations are considered by both the medical profession and the general public to be entirely "safe" and without mortality or morbidity; furthermore, this study is important since in the United States most tonsil and adenoidectomies are performed in communities of this size or smaller.

There is no criticism intended or implied, of the physicians who cared for the cases described in this article, and there is no question that the state of their practice measured well up to that of the community at the time of the occurrences here reported.

Obviously, patients expecting to undergo this operation, as a minimum, should have a well taken history, physical examination, urinalysis and determination of the hemoglobin. Should there be any question of their physical condition, they should also be examined by a pediatrician or internist. Adults should have a chest survey X-ray.

An inquiry into the bleeding habits should be made in each case. This may be confirmed by noting healed wounds and scratches and one or more empty tooth sockets. Determination of the bleeding and coagulation time and platelet counts

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should be reserved for those cases toward which history points the finger of suspicion. Negative examinations are not always dependable.

Mrs. T., now aged 52, had a tonsillectomy 25 years ago. She had a definite history of excessive bleeding after tooth extraction on three different occasions. At three different examinations prior to her tonsillectomy her blood and platelet counts were within normal limits, as was her coagulation and bleeding time. For the three weeks subsequent to her tonsillectomy I spent most of my time stopping bleeding from her tonsillar fossae. As years went by she was hospitalized a number of times, subsequent to dental extractions, for bleeding which demanded transfusions. Sometime later she had a hysterectomy because of excessive flowing at her periods, and eventually had her spleen removed. Other than anemia following her bleeding spells, her blood showed no abnormality.

At the beginning of this 30-year period two children operated on by two different general practitioners who did no other surgery than an occasional tonsil and adenoidectomy, circumcision, or opening an abscess, died of hemorrhage that was not controlled at the time of operation. A sponge was sewed into the tonsillar fossa in one and a tonsil hemostatic clamp that made pressure in the tonsillar fossa and on the outside of the neck was used in the other. These patients were operated on in the sitting position under general anesthesia. No suction machines were available in the Portland hospitals at this time.

This raises the question: "Who should do tonsil and adenoidectomies?" Those physicians who have passed the Boards in Otolaryngology should be qualified; otherwise it is up to the conscience of the individual physician and the judgment of the staff or board of the hospital at which he operates. It would seem that at the present time all interns on a rotating service have the opportunity to learn the technique and the problems involved in the operation of tonsil and adenoidectomy.

It should be impressed again and again on all physicians learning to do tonsil and adenoidectomies that because the chance for mortalities and morbidities to occur is small, their responsibilities for meticulous care for the safety of the patient is greater. Most patients undergoing this operation are small children. Within the last 30 years the average has dropped from eight to ten years to three or four, and these children, whether service or private, are some parents' precious offspring.

Tonsil and adenoidectomies are one of the few surgical procedures in which the wounds are universally left wide open in an infected field. The control of hemorrhage, immediate or secondary, is surgical. It cannot be accomplished by the application of hemostatic sera, solutions of silver nitrate or persulfate of iron. The bleeding point must be found and tied about a hemostat with the fingers, with a slip knot or with a suture, and the bleeding stopped.

Postoperative bleeding after a tonsil and adenoidectomy is possible up to the eighth or ninth day. It happens most frequently in the first 24 hours. Watchful care in this period is the duty of the nursing staff who should contact the surgeon immediately when postoperative bleeding occurs or is suspected. Fresh blood from the nose or mouth and vomiting of blood and blood clots are obvious. The patient's color, the quality and rate of the pulse and respiration should be observed at hourly intervals at least for the first 12 hours.

If any post-tonsil and adenoidectomy bleeding occurs, get the patient's hemoglobin at once; if it is below 60 per cent give the patient an appropriate amount of blood.

The surgeon should seek the origin of the bleeding. There is almost always a clot in the fossa involved, or in the adenoid region, but bleeding may come from a severed uvula or from a nose bleed. Occasionally bleeding may be stopped by removal of the clot and making pressure with a sponge wet with 1-1000 adrenalin, but it is usually better to reanesthetize the patient in the operating room, clean out the clots, and find and tie the bleeding point. Bleeding may come from a partially detached bit of adenoid which should be removed. Retraction of the palate may reveal the bleeding point. It may be safer to insert a post-nasal pack.

I have had two instances in which the blade of a LaForce adenotome passed beyond the basket and caused bleeding from the back of the septum.

The following case history shows the necessity of having the hemoglobin estimated after severe postoperative bleeding:

A boy, age six, was operated on by a skillful general practitioner and surgeon. Throughout the day he had bouts of postoperative bleeding. I saw him in consultation at about 8 p.m., and thoroughly examined his tonsillar fossae and adenoid region while he was under a general anesthesia; after he had vomited up one of those terrifying placentas of blood clots, there was no bleeding. His general condition was good, although he was pale. He died later that night from surgical shock. I believe that he would have been living now if his hemoglobin had been taken, and he was given an adequate transfusion.

The case history cited below illustrates the need for competent postoperative nursing care to detect concealed bleeding:

A child, age six, was operated on by a competent otolaryngologist. She was returned to the ward in excellent condition, and had no evidence of bleeding when visited in the afternoon. During the night she vomited a large amount of blood and died. An autopsy showed her intestinal tract filled with clots.

Sudden death following tonsil and adenoidectomy was formerly considered to be due to status thymo-lymphaticus. The thymus was presumed to swell and obstruct the airways. Studies by Mosher and others have proved that this cannot occur.

Death in the following two patients could have been due to a blood clot from the adenoid region occluding the glottis which might have slid into the pharynx before autospy. It is probable, however, that death occurred from cardiac arrest or ventricular fibrillation. These cases suggest that following a tonsil and adenoidectomy, the patient be kept in the operating room until he has reacted from his anesthetic and then be accompanied to the ward or room by an anesthetist, or preferably taken to a recovery ward equipped with an anesthetist's Laryngoscope, suction machine, and having an attendant trained in anesthesia.

A young male adult, was operated on by a careful general practitioner and surgeon. The patient left the operating room in excellent condition and was being taken up six floors to the ward. Either on the elevator or as he was being taken to the ward, he expired.

A six or seven-year-old child was operated on by a skillful otolaryngologist. The patient left the operating room in excellent condition although the anesthesia had been stormy, and was moved to a ward on the next floor. Just as he was being placed in bed he expired.

A two-year-old child had cardiac arrest just as the tonsil and adenoidectomy was completed. He was operated on by a competent otolaryngologist. Anesthesia had been induced by Vinethane followed by drop ether. The course of the anesthesia was not smooth. Two competent general surgeons immediately opened the chest and massaged the heart. The child lived twelve hours.

Hypoxia of even a mild nature may lead to increased myocardial irritability with the possibility of ventricular fibrillation, cardiac standstill and death. The preoperative use of atropine may protect the heart from vagal stimulation.

Should cardiac arrest occur, the left pleural cavity should be entered by a long incision, in the fifth intercostal space, the costal cartilages above and below may be cut, the hand introduced and the heart compressed at the rate of the operator's pulse.

Two deaths from anesthesia occurred in children at a small hospital primarily devoted to orthopedic cases but in which occasional tonsil and adenoidectomies were performed. The nurse anesthetist was capable of giving adequate ether anesthesia for such cases providing the surgeon kept careful check on the depth of the anesthesia. She was accustomed to sitting at the head of the table, having the operator on the patient's right, and have him control the tongue with a tongue depressor. Motor driven ether vapor was rarely used in this hospital. The otolaryngologist had just completed his training at one of the larger centers where many tonsil and adenoidectomies were performed and where the anesthetists were entirely accustomed to the demands of anesthesia for this procedure. He used a Davis mouth gag with a fixed tongue depressor and sat at the head of the table.

Anesthetists tend to forget that the best and safest anesthesia for tonsil and adenoidectomy in children is well given drop ether. I believe it would be well if every otolaryngological resident spent at least three months in the Department of Anesthesia, particularly, giving tonsil and adenoidectomy anesthesia. The older otolaryngologists watched the depth of anesthesia as carefully as did the anesthetists, for the responsibility for the patient's life was and is theirs.

Preoperative sedation in children with depressant drugs in sufficient doses to insure their arrival in the operating room in tranquil state may cause prolonged postoperative sleep or depression. Gas oxygen or Vinethane gives safe and not too unpleasant induction to drop ether.

Intratracheal anesthesia may be used for adult tonsillectomies done under general anesthesia, if its advantage outweighs the possibility of laryngeal damage. It does not prevent access of blood and mucous to the tracheo-bronchial tree unless the intratracheal tube has an inflatable cuff which increases traumatic risk.

A junior otolaryngologist felt that intratracheal anesthesia would be ideal to use in children when teaching tonsil and adenoidectomy to an intern. The head of the Department of Anesthesia intubated one child and everything went well. The following week a nurse anesthetist had difficulty in tubing a child. A tracheotomy was necessary in the afternoon. Having performed some 70 or 80 tracheotomies for various causes, I have a great respect for the ability of a child's larynx to become edematous and cause an urgent dyspnea.

I have performed tonsil and adenoidectomies with gas oxygen alone, with cyclopropane, and with sodium pentothal, having first anesthetized the larynx, but I believe that drop ether or drop ether supplemented by ether vapor driven by a foot bellows is the safest.

In local anesthesia a death occasionally occurs from accidental substitution of a solution of cocaine for novocaine. Cocaine solutions should be colored, and be in charge of the Department of Anesthesia in the operating room. Novocaine with adrenalin in ampules is almost foolproof, but there are exceptions.

Mrs. X., age 32, was scheduled for a tonsillectomy under local anesthesia. The attending nurse had just come into my employ and had never worked at the hospital in which the operation was to be performed. I noted that the ampule was different than usual and received three affirmative answers that it was 1 per cent novocaine with adrenalin. After I had injected it, I was told that it was Sodium Diodrast. It made a good anesthesia and the recovery was uneventful.

Post-tonsillectomy lung abscesses were formerly considered to occur about once in five or six thousand cases. Seven have occurred in this area, all in the first half of the 30-year period. Six followed general and one a local tonsillectomy; they all occurred in young adults. One underwent external surgery, the others cleared up with bronchoscopic drainage. Blood and mucous has been found in the trachea after both local and general tonsillectomies.

Palpable fremitus from mucous can be noted in the chests of many children when under anesthesia. One school of thought believes that lung abscesses come from infected emboli given off from the tonsillar fossae whose shapes continually change with the muscular action of the pharynx. Another feels that they are caused by inspiration of infected material when conditions are right. *A head low position with adequate use of suction minimizes aspiration.*

Foreign bodies occasionally lodge in the tracheobronchial tree during a tonsil and adenoidectomy. Prior to a general anesthesia the mouth should be searched for not only artificial dentures but also gum and other foreign substances. Loose teeth should be noted. The operator also should inspect his instruments for small loose parts. X-rays taken some two months after tonsil and adenoidectomies revealed teeth in the tracheo-bronchial trees of two children, both of whom were operated on by competent otolaryngologists. In neither of these cases was the loss of a decayed crown or of a deciduous incisor noted by the operator or anesthetist immediately after the operation. They could have been aspirated at a later date, but the presumption is that they were aspirated at the time of the operation, which was done in the prone or dorsal position. This again suggests the advantage of the low head.

A piece of rubber tubing covering the dental surface of a side mouth gag was aspirated. This type of gag is rarely used today.

Pieces of tonsil and adenoids have been aspirated. The use of a LaForce type of adenotome, frequent aspiration, and unremitting care should make such accidents unusual.

I have not adequate check on the number of cases of postoperative bleeding, bronchitis, pneumonia, infectious disease, acute otitis media, pyelitis, nephritis, and rheumatic flare-ups that have followed these operations. There have not been any deep abscesses following local tonsillectomy. One patient had a broken hamular process of his palate, another operated on by a general practitioner received an injury to the lingual nerve when stopping bleeding which caused a loss of taste and sensation on one-half of the tongue, much to the patient's distress.

Asthma and allergy will not be cured by the removal of tonsils and adenoids. Asthma *per se* is not a contra-indication to ether anesthesia as ether is a bronchial dilator.

A boy, age six, who had severe asthmatic attacks, also had tonsils that all but met in the midline and marked difficulty breathing through his nose. I felt that the child would be safer in his asthmatic attacks with his tonsils and adenoids out. He was operated on, and it was my intention to keep him in the hospital for a week, but after four days with absolutely no asthma, his mother insisted upon taking him home to a neighboring town. Two days later he had a severe asthmatic attack and died. I do not feel that the operation had anything to do with his death.

One mild case of poliomyelitis with a quickly clearing paralysis in a leg occurred three weeks after a tonsil and adenoidectomy, which was performed in May, when there had been no other cases reported in this part of the State. Another patient developed poliomyelitis three weeks after a scheduled tonsil and adenoidectomy, which was not done.

SUMMARY AND RECOMMENDATION.

Nine deaths, four from hemorrhage, two at the time of operation and two from postoperative bleeding, three cardiac arrests, two anesthetic deaths, seven lung abscesses, three foreign bodies and a number of minor morbidities followed 20,000 tonsil and adenoidectomies performed by general practitioners and otolaryngologists in a 20-year period in a community of 100,000 people.

It is recommended: that interns on rotating services be taught to perform tonsil and adenoidectomies; that residents in otolaryngology spend at least three months in the Department of Anesthesia; that drop ether is the safest anesthetic for tonsil and adenoidectomy; that solutions of cocaine in the operating room be in charge of the Department of Anesthesia; that atropine be given preoperatively; that all patients having postoperative bleeding have a hemoglobin determination and have an adequate transfusion if their hemoglobin is below 60 per cent; that patients be operated on in a head low position; that the patient be kept in the operating room until he has reacted from his anesthesia and be accompanied to the ward, room, or recovery ward by an anesthetist; that close postoperative watch be kept by the nursing staff for bleeding or vomiting of blood, and that the pulse, respiration, and color be noted at hourly intervals for the first twelve hours; that intratracheal anesthesia be limited to adults; that no preoperative sedation with depressant drugs be used in children; that if cardiac arrest occurs, the chest be opened and the heart massaged; that allergy *per se* is not a contra-indication to tonsil and adenoidectomy; and that the possible relationship of poliomyelitis to tonsil and adenoidectomy be further studied.

FIFTY-SECOND CONGRESS FRANCAIS d'OTO-RHINO-LARYNGOLOGIE.

The 52nd Congrèss Francais d'Oto-rhino-laryngologie will meet October 18 to 22 in the Grand Amphithéâtre of the Faculté of Médecine, Paris, under the presidency of Professeur J. Terracol, of Paris, and honorary president Professeur Lèon Binet, member of the Institut, Doyen of the Faculté of Médecine of Paris. A number of interesting papers will be given, among them, "La Thérapeutique par les ultra sons en Oto-rhino-laryngologie," by Professeur Portmann, with MM. Michel Portmann and Louis Barbe, as collaborators; "De la greffe cutanée libre en chirurgie otologique," by MM. Ombrédanne, Clerc, and Poncet.

AN EXPERIMENTAL STUDY OF THE SMALL BLOOD VESSELS OF THE SPIRAL LIGAMENT AND STRIA VASCULARIS OF LIVING GUINEA PIGS DURING ANAPHYLAXIS.*†‡

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INTRODUCTION.

The purpose of this paper is to describe a series of microscopic observations made upon the walls and intravascular cellular elements of the arterioles, capillaries, and venules of the spiral ligament and stria vascularis of living, anesthetized guinea pigs during anaphylaxis. No one has previously reported such a series of observations.

Several investigators, however, have studied small blood vessels in other tissues of living animals undergoing anaphylaxis. Insertion of transparent chambers into the auricles of the ears of rabbits enabled Abell and Schenck[1] to study arterioles, capillaries, and venules. During anaphylaxis contraction of arterioles, adherence of leucocytes to vessels' walls, emigration of leucocytes through capillary and venular walls, and blocking of capillary blood flow by white blood cell emboli were observed.

In a similar series of experiments Ebert and Wissler[2] noted sticking of leucocytes to arteriolar endothelium, swelling of arteriolar endothelium, localized constrictions and dilatations of arterioles, focal swelling and proliferation of venular endo-

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† From the Winthrop Foundation of the Massachusetts Eye and Ear Infirmary and the Allergy Laboratory of the Massachusetts General Hospital, Boston, Massachusetts.

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thelium, venular dilatation, red cell aggregates, platelet and white blood cell emboli and thrombi.

Thromboses were seen forming in retinal veins by DeMuro and Focosi[3] when they inoculated the vitreus of sensitized rabbits with the specific antigen.

Recently Burrage and Irwin[4,5] have reported changes in the small blood vessels of the livers and lungs of living mammals during the anaphylactic reaction. Contraction of the outlet sphincters of liver sinusoids resulted in storage of blood in the liver. No functioning hepatic arterioles were observed, and linear blood flow stopped in central venules.

Contraction of both pulmonary arterioles and venules was noted when death quickly followed the intravenous injection of the specific antigen. When death did not occur until five minutes or more after the shocking dose of antigen, dilatation pulmonary venules, contraction of arterioles, no open capillaries, aggregates of erythrocytes, formation of emboli and thrombi and leakage of red blood cells from small venules and capillaries were observed.

These experiments as a group at least indicate that changes in small blood vessels do occur when animals undergo anaphylaxis. These small vessels, furthermore, have an important role to play in the physiology of each tissue studied. Changes in them may well be of paramount importance.

MATERIALS AND METHODS.

Over 200 healthy guinea pigs weighing 200 to 1,000 grams were used in these experiments. Observations were made in 57 of these animals in which the blood vessels appeared normal and intravascular cellular elements formed no emboli or red cell aggregates. Each animal was sensitized by two 0.5 ml. intraperitoneal injections of 1-10 solution of crude, sterile egg white five days apart. No animal was presented with a shocking dose until 21 days or more after the initial dose.

In a sensitized and anesthetized animal the spiral ligament and stria vascularis were exposed according to the method described by Weille and associates.[6] The anesthesia (sodium pentobarbital 45 mg. per kg, of body weight) was a principal

dachrome was used, and 24 frames per second when Super X was used. As soon as the shocking dose was given, the animal began to convulse. This required frequent refocusing of the optical system.

OBSERVATIONS.

Vessels studied during these experiments included: the arterioles, the arteriovenous anastomoses, the capillaries, and the venules of the spiral ligament, as well as the capillary network of the stria vascularis. To be considered an arteriole of the spiral ligament a blood vessel had to fulfill certain qualifications. The vessel had to be in the spiral ligament, and it had to take the shape of a cone with the direction of linear blood flow going from the larger end of the cone toward the smaller. In general no part of any arteriole had a diameter greater than 60 microns.

The capillaries appeared in the spiral ligament as an intricate network of dividing and anastomosing cylinders receiving blood from arterioles. The arteriovenous anastomoses were vessels in the spiral ligament. They started off as branches of an arteriole and emptied directly into a venule with no intervening capillary network. At no point did such anastomoses look like cylinders. In the area of pigment cells were found the capillaries of the stria vascularis. These capillaries also formed a network of branching and anastomosing cylinders, and they too emptied into the venules of the spiral ligament. These venules took the form of cones with the direction of linear blood flow from the part of the vessel with the smaller diameter toward that with the larger diameter. Some of these venules ran horizontal to the field, and these in turn drained into venules perpendicular to the field.

During anaphylactic shock certain changes both in the walls of these vessels as well as in the intravascular cellular elements were noted. These changes were by no means consistent, but were more similar when they were compared in animals with the same degree of anaphylaxis. The degrees of anaphylaxis can be divided into three groups; a few animals died within two minutes after receiving the shocking antigen; however, most did not die until five or ten minutes and a few recovered. Obviously there is considerable overlap, and these groups are not clear-cut.

Changes in the calibers of vessels were marked when a sensitized guinea pig died quickly after the shocking dose of antigen. Arterioles contracted. Indeed contraction often was so marked that arterioles were not observed. Figure 1 shows a field with an arteriole of the spiral ligament before anaphylaxis, and Fig. 2 shows the same field during anaphylaxis. Similar behavior of the arteriolar part of arteriovenous anastomoses was observed. Venules contracted, but in only two experiments did they disappear from view. Capillaries of the spiral ligament and those of the stria vascularis were never well visualized because of limitation of magnification. When either set of capillaries were observed both before and during

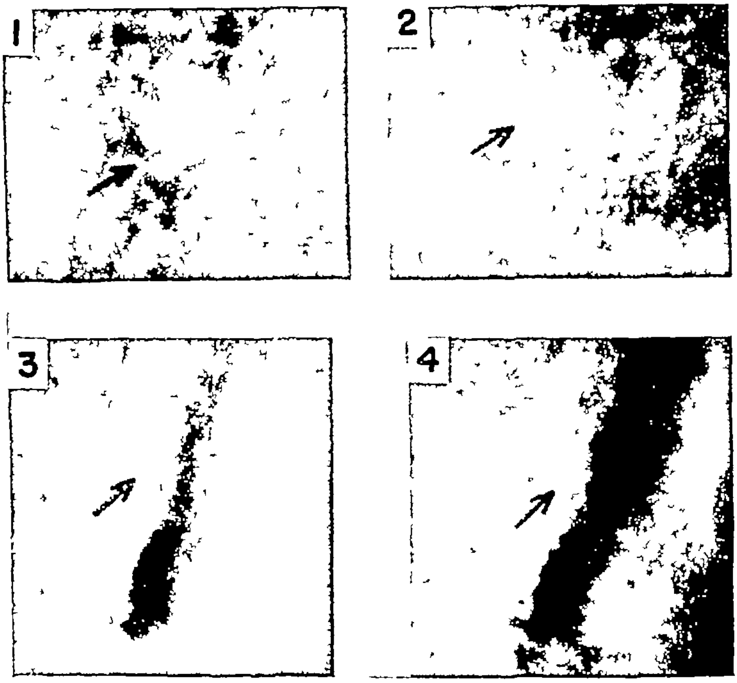

Plate One Fig 1—Arrow Points to arteriole before anaphylaxis Fig 2—Arrow points to same arteriole as in Fig 1 after anaphylaxis Fig 3—Arrow points to a venule before anaphylaxis Fig 4—Arrow points to same venule as in Fig 3 after anaphylaxis

anaphylaxis, the capillaries tended to disappear. Since the walls of capillaries were never well outlined, the apparent disappearance could be due to cessation of blood flow because of arteriolar contraction. Emboli and thrombi were not seen when the reaction progressed rapidly to death.

Usually the sensitized guinea pig did not die until five to ten minutes after the shocking dose of the specific antigen. Again the arterioles contracted, but rarely did they contract tightly shut. The arteriolar parts of arteriovenous anastomoses also contracted. Venules could contract slightly, but the general pattern was dilatation. Figure 3 shows a field with venules before anaphylaxis, and Fig. 4 shows the same venules dilated during anaphylaxis.

The venular portions of the arteriovenous anastomoses followed the venular action. Capillaries of the spiral ligament and stria vascularis disappeared. At least they could not be visualized. Masses highly refractile to light appeared on both the arteriolar and venular sides of the circulation. Aggregates of erythrocytes were also seen. Often blockage of a venule or arteriole by one of these highly refractile masses or one of the red cell aggregates was observed. Experiment No. 1 illustrates many of the above points.

EXPERIMENT NO. 1

Male Guinea Pig—Weight 1,050 grams; egg white sensitive.

Date—August 25, 1953.

10:00 A.M.—42 mg. of sodium pentobarbital intraperitoneally.

10:30 A.M.—Surgery started.

2:30 P. M.—Observations started.

2:35 P. M.—Two branching arterioles; rapid linear blood flow. Two arteriovenous anastomoses; rapid linear blood flow. Three venules running horizontal to field; rapid linear blood flow. Several venules running vertical to field; rapid linear blood flow. Actively flowing capillaries of stria vascularis and spiral ligament.

2:40 P.M.—0.1 ml. egg white 1-10 intravenously.

2:41 P.M.—Contraction of arterioles and arteriolar parts of arteriovenous anastomoses; venules dilating.

2:44 P.M.—Irregular shaped emboli highly refractile to light observed in arterio-venous anastomoses and venules as well as aggregates of erythrocytes. These emboli stretched the arteriolar walls as they passed through, but the walls returned to constricted phase after emboli passed.

2:46 P.M.—Arterioles contracted; no linear blood flow.

2:47 P.M.—Emboli stuck in venules, blocking flow.

2:48 P.M.—Capillaries not visualized.

2:51 P.M.—No apparent linear blood flow in any vessel.

2:52 P.M.—Guinea pig died.

Anaphylaxis did not prove to be fatal in a few guinea pigs. The arterioles and arteriolar part of arteriovenous anastomoses constricted. Venules dilated. The capillaries either showed no change or disappeared from view. Emboli highly refractile to light were seen in all vessels, and aggregates of erythrocytes were found in all vessels. Both types of emboli appeared more evident on the venous side of the circulation. Frequently emboli would plug the lumen of small arterioles and venules. These emboli might, therefore, be classified as thrombi. If the animal lived, the circulation returned to normal. Emboli disappeared and arterioles and venules returned to their original calibers. In no instance, however, did the thrombi ever disappear. It must be pointed out that no field was visualized for more than four hours after the shocking dose. Experiment No. 2 will point out several of these factors.

EXPERIMENT NO. 2

Male Guinea Pig—Weight 975 grams; egg white sensitive.
Date—August 26, 1953.

9:00 A.M.—33 mg. of sodium pentobarbital intraperitoneally.

9:30 A.M.—Surgery started.

12 noon—Observations started. Arteriole: rapid linear blood flow. Arteriovenous anastomoses; rapid linear blood

flow. Two venules running horizontal to field; rapid linear blood flow. One venule running vertical to field; rapid linear blood flow. Capillaries of stria vascularis; network.

12:15 P.M.—0.3 ml. egg white intraperitoneally.

12:16 P.M.—Constriction of arterioles and arteriolar part of arteriovenous anastomoses. Venules dilated. Capillaries: no change. Emboli highly refractile to light in all vessels. Aggregates of red cells in all vessels.

12:17 P.M.—Flow in all vessels. Emboli in all types of vessels under observation.

12:18 P.M.—Emboli have plugged one arteriole and one arteriovenous anastomsis (*i.e.* thrombi).

12:21 P.M.—Arterioles dilating. Flow in all vessels. Fewer emboli.

12:40 P.M.—Vessels as at start of experiment before shocking dose.

3:00 P.M.—No emboli. Thrombus still in one arteriole, and in one arteriovenous anastomosis.

DISCUSSION.

In any series of experiments certain experimental conditions are introduced which may well effect end results. It was necessary to anesthetize deeply each guinea pig with sodium pentobarbital. Unfortunately, with sodium pentobarbital the margin between adequate narcosis and death was narrow. Sodium pentobarbital does affect to some extent small blood vessels, but no better agent is available.

Operative procedures, used to expose the cochlea and to fenestrate one of its turns, were extensive. If the spiral ligament or its blood vessels were damaged, the guinea pig was discarded. Thus anesthesia and surgical trauma accounted for most of the failures. Once a successful fenestra was made, it was imperative to keep the exposed tissue moist and at normal temperature.

The amount of light needed for observation was large. This light itself was responsible for considerable heat. The problems of heat and moisture were controlled in part by a gentle flow of Mammalian Ringer's which emitted from the very tip of the quartz rod at a constant rate and temperature. Controls to date are crude and much remains to be done in this phase.

The problem of magnification remains. Objectives of 20x to 90x have short working distances, and are so large in diameter that they can not be placed near the fenestra because of the narrowness of the bony vault about the cochlea. Until adequate means of magnification are available, accurate observations upon the capillaries must wait.

In spite of inherent difficulties over fifty successful preparations were studied. All observations were made on these animals. The blood vessels described under observations were found in each animal. The intravascular cellular elements formed no emboli or thrombi until anaphylaxis was induced.

Certain changes both in the calibers of the small blood vessels of the spiral ligament and in the intravascular cellular elements occurred consistently during anaphylaxis which is a general systemic reaction depending on antigens and antibodies. During anaphylaxis the main changes in calibers of vessels appeared to be constriction of the arterioles of the spiral ligament and dilatation of the venules.

At times the arterioles constricted tightly shut. Since these vessels quickly returned to normal size if the animal survived anaphylaxis, it is difficult to determine how important such constriction and dilatation might be. The formation of emboli and thrombi within the vessels appears to be of more importance. Even though most of the emboli disappeared once an animal survived, thrombi plugging certain of these vessels did not disappear. Such plugged vessels would be unable to perform their usual function. Surrounding tissue might consequently be damaged.

SUMMARY.

1. Anesthetized guinea pigs sensitive to egg white can die within 2 or 3 minutes after the shocking dose of antigen. The changes noted in the blood vessels of the spiral ligament included constriction of arterioles and initial constriction followed by dilatation of venules.

2. When death did not occur until several minutes after the shocking dose, beside constriction of arterioles and dilatation of venules of the spiral ligament, emboli and thrombi were observed in both arterioles and venules.

3. When the guinea pig recovered from anaphylaxis, thrombi in the vessels did not disappear.

4. The phenomena observed in the inner ear of the guinea pig during anaphylaxis may or may not apply to other mammals, but they are of interest since such observations in the labyrinth have not been previously reported.

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ANNOUNCEMENT.

An introductory course in Reconstructive Surgery of the Septum and External Nasal Pyramid will be given, under the direction of Dr. Maurice Cottle, March 19 through 26, 1955 at the University of Oregon Medical School. This will be sponsored by the Department of Otolaryngology.

THE SURGICAL TREATMENT OF THE ATRESIA AURIS CONGENITA; A CLINICAL AND HISTOLOGICAL REPORT.*†

By Professor L. RUEDI
Zurich, Switzerland.

With the aid of fenestration technique and the use of antibiotics, the prospects of surgical treatment of congenital atresia of the ear have become more favorable. Of course, the opinions concerning the indications to operate and the technique to be used are still not quite settled. M. Ombrédanne operates preferably on cases of one-sided atresia in order to relieve these mentally depressed patients and to obtain a reserve of hearing. He creates a new meatus, and in the same stage of the operation the horizontal semicircular canal is fenestrated. G. L. Pattee performs the operation primarily on patients who are bilaterally affected. After making a new external meatus the incus is removed. By this method the stapes should become mobile, and the hearing should improve as with a successful fenestration. R. F. Guilford and George E. Shambaugh refrain from removing the incus where there is only a slight alteration of the ossicles and the incudo-malleolar articulation seems functionally normal. The new meatus ends in a skin flap which seals off the tympanic cavity and covers the auditory ossicles. In bilaterally affected patients, DeGraaf Woodman recommends a two-stage operation as suggested by F. Altmann. In the first stage a new meatus is made, and the chain of auditory ossicles is disconnected. If after the first stage the hearing gain is insufficient, a fenestration of the horizontal canal will follow in a second operation.

Our experience with the operative treatment of congenital atresia of the ear, collected since 1948, concerns four female and nine male patients, ranging in age from 7 - 39 years

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† From the Oto-Rhino-Laryngological Clinic of the University of Zürich.

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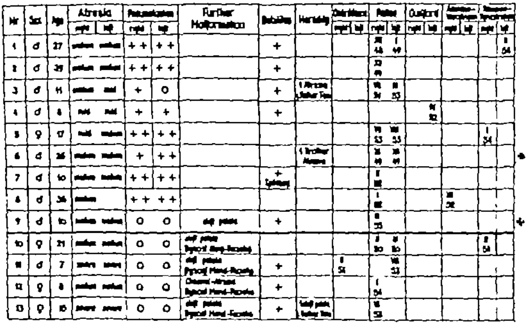
(see Fig. 1). Twelve of these patients were afflicted bilaterally and all of them had intact inner ear function, thereby fulfilling the indication to operate. One patient with one-sided atresia had to be operated upon because a perception deafness gradually developed in the second previously normal ear. In six patients both ears and in seven patients, one ear, were operated. The best cosmetic results obtained by operation are in our opinion surpassed by auricle prosthesis of plastic material; therefore, this aspect of the atresia problem will not be treated in the current discussion.



Nr	Sex	Age	Atresia		Pneumatization		Further Malformation	[illegible]	Heredity	[illegible]		[illegible]		[illegible]		[illegible]		[illegible]		
			right	left	right	left				right	left	right	left	right	left	right	left	right	left	
1	♂	27	medium	medium	+ +	+ +		+				[illegible] 44	[illegible] 49						[illegible] 54	
2	♂	25	medium	medium	+ +	+ +		+				[illegible] [illegible]								
3	♂	11	medium	mild	+	O		+	1 [illegible] [illegible]			[illegible] 54	[illegible] 53							
4	♂	8	mild	mild	+	+		+							[illegible] 52					
5	♀	17	mild	medium	+ +	+ +						[illegible] 53	[illegible] 53					[illegible] 54		
6	♂	26	medium	medium	+	+ +			1 Brother [illegible]			[illegible] 49	[illegible] 49							+
7	♂	10	medium	medium	+ +	+ +		+ [illegible]				[illegible] [illegible]								
8	♂	36	medium		+ +	+ +						[illegible] 52				[illegible] 52				
9	♂	10	medium	medium	O	O	cleft palate	+				[illegible] 53								+
10	♀	21	medium	medium	O	O	cleft palate Typical [illegible]					[illegible] 50	[illegible] 50					[illegible] 54		
11	♂	7	severe	severe	O	O	cleft palate Typical Hand-[illegible]	+		[illegible] 51			[illegible] 53							
12	♀	8	medium	medium	O	O	Choanal-Atresia Typical Hand-[illegible]	+				[illegible] 54								
13	♀	10	severe	severe	O	O	cleft palate Typical Hand-[illegible]	+	[illegible] [illegible]			[illegible] 53								

Fig. 1. Schedule.

As to the severity of malformation our cases can be subdivided, according to H. Marx and F. Altmann, by means of the ear status, roentgenological examination and operative findings, in the following manner:

Four ears show atresia of mild degree.

Seventeen ears show atresia of medium degree.

Four ears show atresia of severe degree.

The characteristic anatomical changes in these various degrees of malformation have been excellently described by F. Altmann.

In a first group of nine patients (above the dotted line) there exists in one case an additional malformation, a cleft palate. In this group, roentgenological examination of the mastoid in eight of nine cases revealed good to very extensive pneumatization. This confirms the observation of F. Altmann that "in almost all cases with hypoplasia of the middle ear, alone or combined with hypoplasia of the tube, there was a mastoid process of normal size, and as a rule with good development of the pneumatic cells."

In the second group of four cases (below the dotted line), in addition to the congenital atresia there was malformations, mainly of the head and, in one case, also of the extremities. In these four cases the mastoid process is almost completely absent, and the pneumatic cells could not be seen either roentgenologically or during the operation. In all four cases of the second group the malformation syndrome known as mandibulo-facial dysostosis as described by Treacher-Collins and by A. Franceschetti and P. Zwahlen, was present.

The Franceschetti-Zwahlen syndrome differs from O. Crouzon's cranio-facial dysostosis in that the former has no malformation of the cranium. F. R. Nager observed two of our cases (Nos. 10 and 13) for many years. Both cases were extensively reported by F. R. Nager and J. P. de Reynier, in the monograph "The Hearing Organ in Congenital Malformation of the Head."

We will, therefore, restrict ourselves to a short description of the changes in the facial part of the skull which were manifest in our four cases, in addition to the atresia of the ear.

Case No. 10 concerns a girl born in 1929. At birth the child showed malformations of both auricles, abnormalities of both thumbs and a cleft palate. The cleft palate was operated upon when the child was two and four years of age. The child began to speak at the age of one and one-half years. Of normal intelligence, she successfully attended several schools. In addition to the congenital atresia, malformed auricles and absence of the meatus, the face (see Fig. 2) shows a slight hypoplasia of the maxilla and symmetrical slanted position of the eyes. The thumbs (see Fig. 3) are absent on both hands. The right arm is rigidly fixed at a right angle and can be supinated and pronated only in a limited manner. Roentgenologically the cranium is normally configurated, the sinuses are small, the zygomatic arches hypoplastic and the maxilla and mandibula are somewhat underdeveloped. In the roentgen picture of the upper extremities there is a synostosis of the radius and ulna in the proximal third. In both hands the thumb is absent and also missing are the phalangea, metacarpal I, multangulum majus and the naviculare. The multangulum majus is fused with the naviculare.

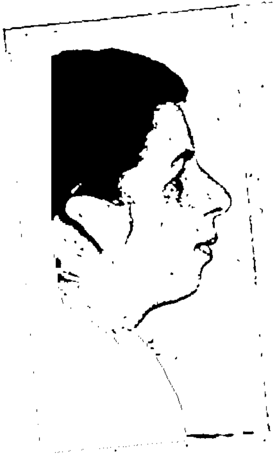


Fig. 2.

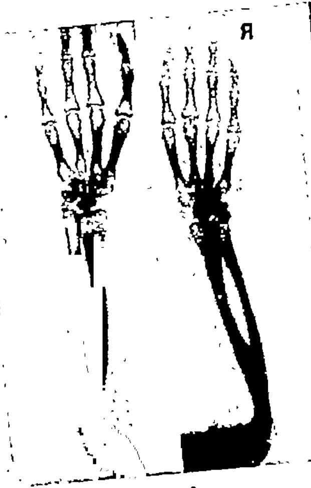


Fig. 3.

Fig. 4.

Fig. 2. Case No. 10. Female, 21 years, Atresia auris congenita with dysostosis akro-facialis.
Fig. 3. Case No. 10. Roentgen Picture of the upper extremities.
Fig. 4. Case No. 13. Female, 16 years, Atresia auris congenita with dysostosis mandibulo facialis.

To summarize there exists a congenital ear atresia, a mandibulo-facial dysostosis, combined with malformation of the extremities.

Case No. 11 concerns a boy born in 1945 with bilaterally malformed auricles, absent meatus, a cleft palate and malformation of the facial skull. The intellectually retarded child began to speak at four years of age. The cleft palate was operated upon in 1952. On the face there may be seen a hypoplasia of the upper and lower jaw and a slanted position of the eyes.

It is again a case of mandibulo-facial dysostosis (A. Franceschetti and P. Zwahlen).

Case No. 12 is a girl born in 1946 afflicted with a congenital atresia of the ear which is restricted bilaterally to the middle ear. The large, protruding auricles are normal. The outer ear canal is present on both sides. On both sides a pale red, translucent ear drum with severely reduced mobility can be seen. Hearing tests show a severe sound conduction deafness on both sides.

In addition there is a choanal atresia on the left side and an underdevelopment of the facial skull showing a mandibulo-facial dysostosis.

Case No. 13 is a girl born in 1937, who at the time of birth showed deformed auricles, absence of the outer ear canals, a cleft palate and a severe deformation of the facial skeleton. At the age of one and one-half years the child could barely speak. At the age of two and four years the cleft palate was operated upon. In Fig. 4 the typical "bird" face can be recognized with a prominent hooked nose, hypoplasia of the mandible, hypoplasia of zygomatic arches, slanted position of the eyes and coloboma of both lower lids. The roentgen picture of the skull shows an internal hydrocephalus with normal sutures compensatory enlargement of the posterior fossa and a steep position of the base of the skull.

Here again a very pronounced case of mandibulo-facial dysostosis is present.

In the four cases of our second group with malformations of the ear and facial skeleton the following abnormalities are found:

(a) a bilateral congenital atresia of the ear of medium to severe degree;

(b) a hypoplasia of the zygomatic arches and mandible;

(c) eye lids slanted down laterally with a peculiar bending of the lateral third of the lower lid and coloboma formation;

(d) cleft palate in three of four cases, in one case a choanal atresia;

(e) Case No. 10 showed a deformation of the upper extremities in addition.

A certain parallel can be drawn to acro-cephalo-syndactyly observed by E. Apert and to cephalo-syndactyly described by A. Vogt, who in 1930 called attention for the first time to the combined occurrence of a cranio-facial dysostosis; that is, Crouzon's disease, with symptoms of Apert's disease. In view of these combined malformations, F. R. Nager has proposed the term acro-facial dysostosis for our Case No. 10. Pathogenetically we must assume a disturbance of the organizer of the trunk as well as of the head.

The intellectual development was clearly retarded in nine of the 13 cases with ear malformation. In the group with uncomplicated atresia (possibly combined with a cleft palate), three out of nine patients are normally intelligent; in the groups of complicated ear and face malformations, only one out of four.

There is as yet very little known as to the cause of ear and skull malformation. F. Altmann has recently mentioned genetic and environmental factors that could damage the organizer of the head in the early embryological state. Two of our patients have blood relatives with congenital malformation of the ears. Cleft palates occurred in the families of three other patients, so that genetic factors would appear to play a role in these cases.

Finally — Fig. 1 gives information concerning the respective surgical therapy. Since we were followers of Pattee's method, 12 of the 19 ears were operated upon in that fashion. [In Case No. 4 only slight alterations of the tympanic cavity and a mobile malleus-incus articulation were found, which is the reason that the ossicles were left intact (modification, Guilford-Shambaugh).] In Case No. 11 with combined ear-face malformation the one-stage method of M. Ombrédanne was used. F. Altmann-De Graaf Woodman's two-stage operation was performed once and tried once, and finally in three late re-examinations, secondary fenestration was done.

Technically, operating on the uncomplicated atresia with good pneumatization of the mastoid presents no special difficulties. The operation is very difficult, however, in cases of mandibulo-facial dysostosis. As a rule there is no mastoid process, the guiding pneumatic cells, the antrum and frequently also the aditus ad antrum are absent. The widely exposed dura of the middle fossa and the sino-dural angle serve as guiding points for locating the middle ear cavity, which is always very small. The middle ear cavity is found by following the exposed dura with the burr in a ventrally-medial direction. The reasons for certain modifications in our surgical procedure can be seen from the critical observation of our early and late results:

Ten ears operated with the Pattee method (see Fig. 5) (Case 4 with Guilford-Shambaugh modification), showed a good to sufficient hearing gain in the immediate post-operative period. The audiograms of uncomplicated atresia are above the dotted line; the audiograms of the complicated cases with ear, face and skull malformations are below the white line.

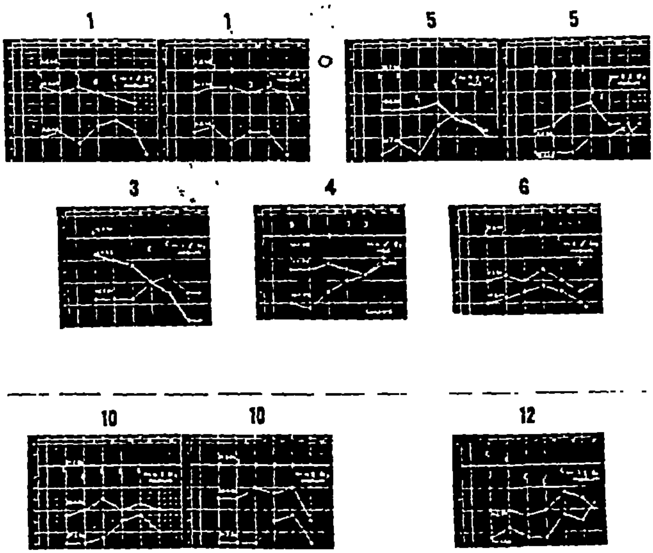


Fig. 5. Audiograms of atresia auris congenita showing good or sufficient hearing gain after the Pattee operation.

Eight ears operated according to Pattee show insufficient or no hearing gain (see Fig. 6). There are eight failures, as compared to 10 early successes of the Pattee operation. The operative failures of cases Nos. 11 and 13, with combined face and skull malformation, can be explained by the extensive *deformity in the ear region.*

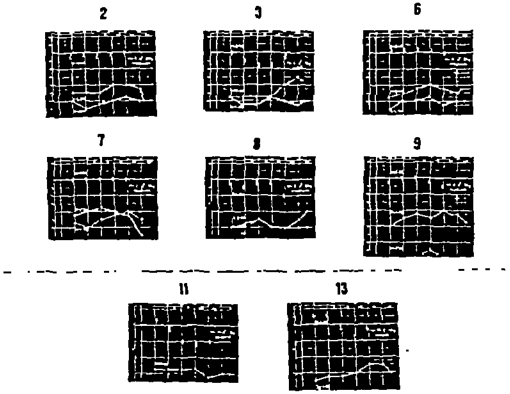


Fig. 6. Audiograms of atresia auris congenita showing insufficient hearing gain after the Pattee operation.

In Case No. 13 (right ear) after hours of searching, a small slit was found. filled with red granulation tissue and rudimentary ossicles. The stapes could not be found. From all appearances the tympanic cavity was present only as a small slit, so the operation was terminated without the formation of a plastic meatus.

In Case No. 11 (left ear) where the antrum and aditus were absent, a very small middle ear cavity was finally found and a rudimentary incus removed from a fold of mucous membrane. Subsequently the exposed middle ear cavity and the new meatus were lined with epithelium in the typical manner by a pedunculated skin flap.

The negative operative results in the uncomplicated atresia (above the dotted line) cannot be conclusively explained by the operative findings.

Two patients of this group (Case No. 6, left ear; Case No. 9, the right) died of causes other than the operation.

Case 6, a gifted young man with a University degree was operated bilaterally, the right ear with sufficient, the left with insufficient, hearing gain. During his first sojourn abroad this patient committed suicide while in a manic depressive attack from which he had been suffering some time. One week before his death he wrote of how wonderful it was to have regained the hearing in his right ear and of the new hope for the future that it gave him.

Case 9 was a debilitated boy of 10 years with bilateral congenital ear atresia and a cleft palate, who presented the following operative findings:

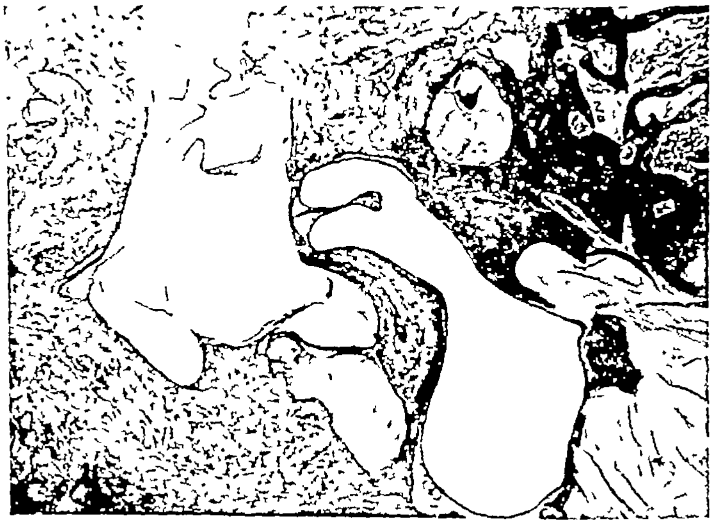


Fig. 7 Case No 9 Male, 10 years, Atresia auris congenita Left temporal bone (not operated) (Vertical serial section) Section through the window-niche

Small mastoid process without pneumatization Large ventral displacement of the sigmoid sinus. Very low middle fossa, consequently very cramped relationship. Finally a very small antrum and a small aditus were opened Connective tissue found in both spaces was removed. An ossicle resembling an incus could then be extracted. The stapes could not be seen. *Formation of a skin flap to line the newly formed external* canal and to close the middle ear in the operative cavity was done.

Post-operatively there was a clear but for practical purposes insufficient hearing gain.

Six weeks following the operation the patient sustained a fatal accident.

Histological examination of the petrous bone.

Left temporal bone (not operated) (vertical serial section). Section through the window. (See Fig. 7). In the lateral wall of the middle ear cavity, the tympanic bone, the tympanic membrane and the external meatus are missing. The resulting defect was partially closed by a downward extension of the squama temporalis. On the other side a bony lamella extends upward from the lateral part of the floor of the middle ear. The defect in the lateral wall is filled with strands of dense connective tissue. The squama is situated very close to the oval window of the inner ear capsule so that the tympanic cavity is extremely low. In this bony bridge the ventrally displaced facial nerve runs. The oval window, the foot plate of the stapes and the ligament are normal. The low tympanic cavity is so small here that the head of the stapes (see Fig. 8) touches the bony wall of the facial canal and is joined to it by connective tissue. Other than the stapes there are no auditory ossicles seen in this slide. The niche of the round window appears completely filled with a loose connective tissue in all sections. The inner ear is normal.

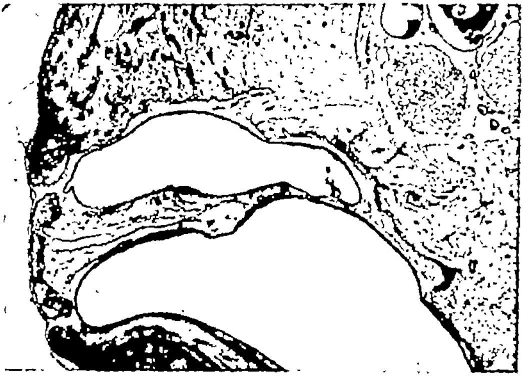


Fig. 8. Case No. 5. Male, 10 years. Atresia auris congenita. Left temporal bone (not operated). Fibrous adhesion between the capitulum stapedis and the bony wall of the facial canal.

Section through the aditus ad antrum: In a fairly wide lumen a remnant of the incus lies in a wide meshwork of connective tissue, without connection to the stapes.

Right temporal bone (Pattee operation) (vertical serial section). Section through the window: (See Fig. 9). In the lateral wall of the middle ear cavity the tympanic bone, the tympanic membrane and the external meatus are missing. The resulting defect is completely filled out by a downward extension of the squama temporalis. The middle ear cavity on this side extends way above the upper edge of the oval window to the widened tegmen tympani. The facial nerve here runs normally, in front of the upper edge of the oval window. The oval window, the foot plate of the stapes and the ligament are normal. On this side also the niche of the round window is filled completely with a loose, partially myxomatous connective tissue.

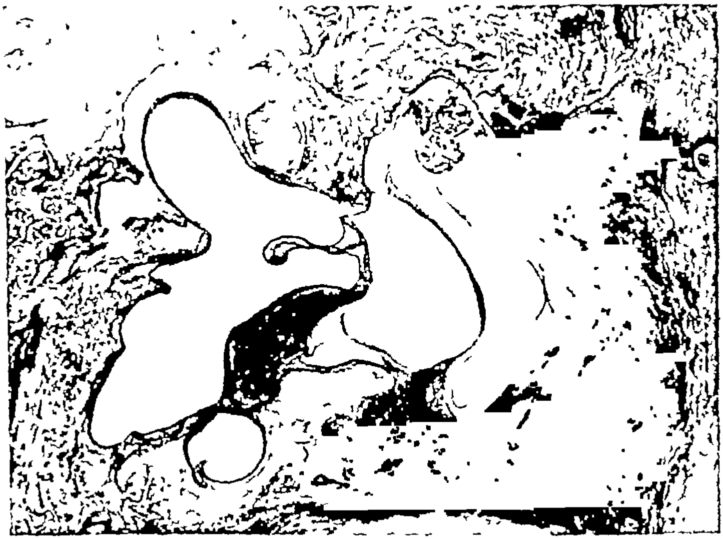


Fig 9 Case No 9. Male, 10 years, Atresia auris congenita, Right temporal bone (Pattee operation) (Vertical serial section). Section through the window-niche

Section through the medial part of the capitulum stapedis: The capitulum is plump and broad and is bound to the bony canal of the facial nerve by a broad band of connective tissue. The narrow lumen of the tympanic cavity is lined with cuboidal epithelium whose intact surface is covered with a layer of inflammatory exudate. Extensive inflammatory infiltrations are found in the loose mucosal connective tissue.

Section through the medial part of the newly formed meatus acusticus externus: The tube of squamous epithelium terminating here shows superficial desquamation. The tunica propria shows inflammatory infiltrations.

Section through the lateral part of the newly formed meatus acusticus externus:

On the basis of similar histological changes, F. Altmann has already called attention to the fact "there may exist a fixation of the stapes by bony bridges to the medial wall or bridges between the auditory ossicles and the medial superior or outer wall which would remain undiagnosed by present methods of clinical examination."

F. Altmann and De Graaf Woodman suggest that the operative failures due to fixation of the stapes or occlusion of the niche of the round window should be fenestrated in a second phase. Since we do our otosclerosis operations in two stages routinely the same approach to the treatment of atresia seems most convenient and suitable.

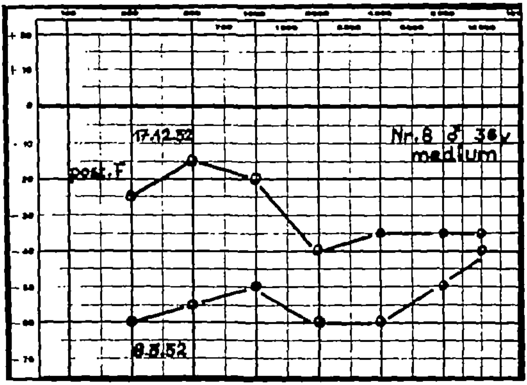


Fig. 10. Case No. 8. Audiogram of May 8, 1952, after Pattee operation. Audiogram of December 17, 1952, after secondary fenestration.

In Case No. 8 (the right ear) was fenestrated 11 months after the Pattee operation which had produced insufficient hearing gain. Two weeks post-op, the audiogram (see Fig. 10) shows a considerable improvement. Three other ears from the group of failures (Cases No. 2, 3 and 7) were suited for secondary fenestration. Two will be operated upon. The third case (Case No. 3) was meanwhile fenestrated in the second ear by Ombrédanne, in a single-stage operation. Unfortunately a planned recheck of both ears could not yet be done.

Longstanding results on the operated atresia, as far as we know, are reported only by George E. Shambaugh; therefore, we have rechecked our patients who after the Pattee operation initially showed a good to sufficient hearing gain post-operatively, with the following results: (see Fig. 11).

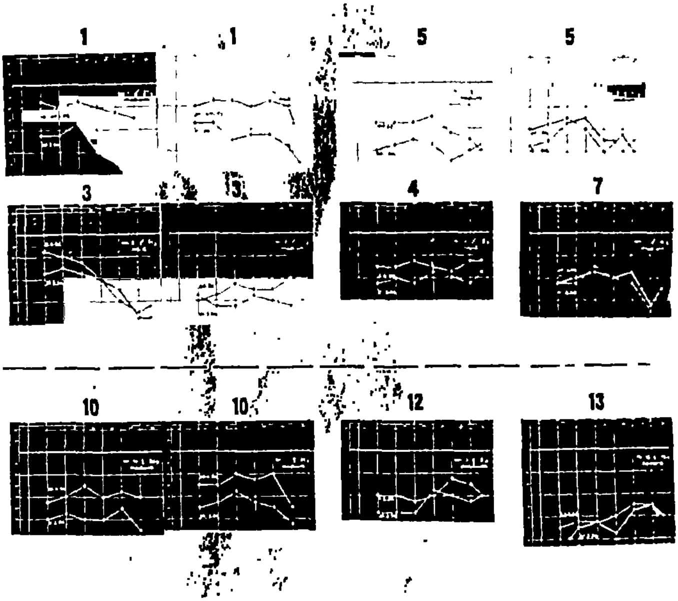


Fig. 11. Audiograms of atresia auris congenita. Longstanding results after the Pattee operation in initially successful cases.

Case No. 1 was operated by the Pattee method bilaterally with a very good hearing gain which lasted three years. Only in the course of the last two years has a hearing loss once again developed. The reason for this rather late developing hearing loss is unknown. The patient had recurrent eczematous irritation of the newly formed external meatus but was regularly treated by a specialist. In Case No. 5, operated bilaterally, the post-operative hearing gain in the right ear decreased gradually in the course of a year, while the left ear remained constant. Gradual hearing loss following an initial post-operative gain also developed in the left ear of Case No. 3, in the left ear of Case No. 4, and in three ears with combined congenital ear atresia and mandibulo-facial dysostosis.

The result of our review is anything but satisfactory. Of the 10 ears operated by the Pattee method with initially good results only two ears maintained the hearing gain after one to five years. One patient in this group died of other causes.

To arrive at some explanation for this late decline of the hearing gain we did a revision operation in Case Nos. 1, 5, 10 and 12, by lifting up the skin flap closing the middle ear. Simultaneously a fenestration of the horizontal semicircular canal was done, where possible, with the following results (see Fig. 12).

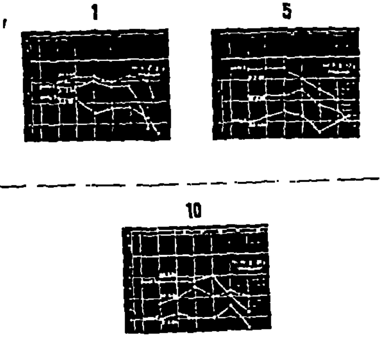


Fig. 12. Audiograms of atresia auris congenita, after revision and secondary fenestration of an initially successful Pattee operation.

In Case No. 1 (the left ear) after lifting off the skin flap the under surface showed a peculiar, gelatinous, layered arrangement of the connective tissue. In the tympanic cavity there were numerous bands of connective tissue which were removed as far as possible. Locating the horizontal semicircular canal was very difficult. A window was made in the dome and covered by a skin flap.

Re-examination and fenestration in this case resulted in a notable hearing gain. The dotted curve shows the hearing gain obtained by the Pattee operation which remained at this level for three years. The drawn out lower curve in the audiogram represents hearing before fenestration. By revision and fenestration the previous hearing gain was restored almost to the original level.

In Case No. 5 (the right ear) granulation tissue was first removed from the posterior part of the newly formed external meatus. After lifting off the skin flap, some new bone formations over the horizontal semicircular canal were found. The tympanic cavity contained newly formed connective tissue which was removed as far as possible. A fenestration could then be done without difficulty and the fenestra was covered with a skin flap.

Ten days following fenestration, hearing was extraordinarily improved. The audiometric curve now runs above the dotted line representing hearing following the Pattee operation.

In Case No. 10 (the right ear) the revision operation was extremely difficult because of the close relationship and the abnormal position of the horizontal semicircular canal, due to the mandibulo-facial dysostosis. The narrow skin canal leading into the middle ear had to be completely removed. It was impossible to see into the small tympanic cavity, partially filled with scar tissue. The horizontal semicircular canal was found dorsally displaced after a three and one-half hours' search in bone, and finally fenestrated on its posterior surface. The window was closed by a finger-like tube of skin.

Thirteen days following fenestration this complicated atresia case also showed a hearing curve exceeding the gain achieved by the Pattee operation alone (the dotted line).

In Case No. 12 (the right ear) the anatomical relationship did not at first appear unsuitable for a revision despite the combination of atresia with dysostosis mandibulo-facialis; since here the malformation was restricted to the tympanic cavity alone (that is, the outer ear), the canal and a small immobile ear drum were present.

After lifting the skin flap, newly developed connective tissue adhesions were found and removed. In four hours of boring it was impossible to locate the horizontal semicircular canal. Finally, under the assumption that the inner ear had been damaged, the search was abandoned. Subsequently this fear was fortunately not substantiated. Despite hours of unsuccessful drilling in the labyrinth the child hears as much following the revision, as before.

In the course of four revisions of initially successful Pattee operations, newly formed connective tissue was found in the middle ear cavity and removed. It is quite possible that this scar tissue formation is partially responsible for the gradually developing hearing loss; however, removal of the scar tissue *alone* is *not* sufficient to regain hearing as is shown in Case No. 12.

A decisive hearing gain is achieved only after fenestration of these cases. As a result of our experiences with the revisions, we are today convinced of the significance of fenestration in the treatment of atresia. We will continue to operate uncomplicated atresia cases with the Pattee method first, whereby the stapes will be located, mobilized and possibly the capitulum stapedis resected. If, however, an insufficient hearing gain is thus achieved, then a secondary fenestration will be done. In those rare cases of congenital atresia combined with skull malformation, the revision, that is secondary fenestration, according to our observations, is technically so difficult and so risky that the patient should not be subjected to the operation. We find ourselves therefore, in a paradoxical situation. Precisely the topographically most difficult cases are not suitable for the simpler two-stage method; so that the complicated atresia should be done by the more difficult one-stage operation.

Accordingly, following an unsuccessful five-stage operation on the left ear, a one-stage fenestration of the right ear was undertaken in Case No. 11.

A severely thickened mucous membrane and deformed auditory ossicle were removed as completely as possible from the extremely small tympanic cavity. The gaping petro-squamosa fissure which lay directly on the tympanic cavity's mucous lining was filled with connective tissue. The tensor tympani muscle was removed. To localize the horizontal semicircular canal the facial nerve was exposed for a short distance and the fenestration was performed in the region of the dome. The window was closed with a pedicled flap of skin and the remaining cavity lined with a Thiersch skin transplantation.

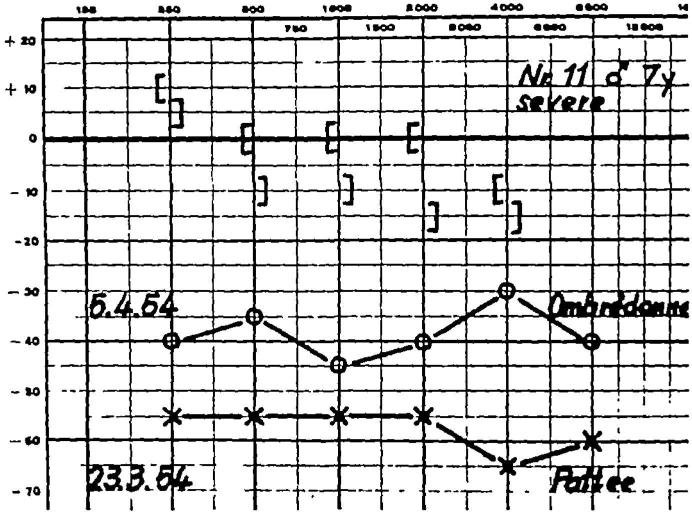


Fig. 13. Audiogram of March 23, 1954, after Pattee operation of the left ear. Audiogram of April 5, 1954, after Ombrédanne operation of the right ear.

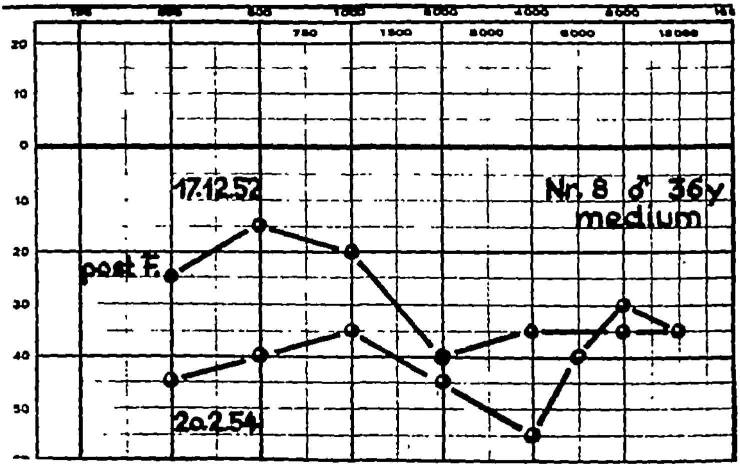


Fig. 14. Audiogram of December 17, 1952, after secondary fenestration. Audiogram of February 20, 1954, showing a hearing loss after the initially successful fenestration.

The audiometric curve (see Fig. 13) of the fenestrated right ear is closely above the Pattee operated left ear.

Finally the important question as to the permanent results of the fenestration treatment of congenital ear atresia presents itself. Unfortunately for this consideration we have only the one test patient. In this (Case No. 8) a decrease of the hearing gains due to fenestration occurred in the course of two years (see Fig. 14). Naturally we cannot draw any conclusions from one such observation. George E. Shambaugh has reported two fenestrated atresia cases with still sufficient hearing improvement three years after the operation; therefore, it seems worthwhile to continue our surgical efforts. It seems important that we also obtain further observation on fenestrated atresia cases.

SUMMARY.

A total of 19 ears in 12 cases of bilateral ear atresia and one case of one-sided atresia, were operated upon. Nine of these cases were uncomplicated atresia, four were atresia combined with mandibulo-facial dysostosis. In 10 out of 18 ears a good to adequate hearing gain was first achieved by the Pattee method. Operative failure could be histologically verified in one case as due to fixation of the stapes. In seven out of 10 Pattee cases the operatively achieved hearing gain was lost in the course of several years. By revision and fenestration the initial hearing gain could be restored or exceeded, in three cases.

For uncomplicated cases of ear atresia the two-stage fenestration operation, combined with stapes mobilization and possible resection in the first phase is recommended. Ear atresia, combined with malformations of the skull, should be fenestrated in a one-stage procedure. The prognosis of the fenestrated atresia cases has yet to be determined on the basis of the material outstanding.

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POST GRADUATE SESSION IN OPHTHALMOLOGY AND OTOLARYNGOLOGY.

A Post Graduate Session in Ophthalmology and Otolaryngology sponsored by the Virginia Society of Ophthalmology and Otolaryngology will meet at the University of Virginia from November 30 to December 3, 1954.

November 30 and December 1, will be devoted to Otolaryngology lectures and December 2 and 3, will be devoted to Ophthalmological subjects. Those interested please contact Dr. Edwin Burton or Dr. G. Slaughter Fitz-Hugh, 104 East Market Street, Charlottesville, Va.

PROGRESSIVE ORAL GANGRENE DUE TO ACATALASEMIA (COLORED MOTION PICTURE).[*][†]

SHIGEO TAKAHARA, M.D.,
(By Invitation),
Chicago, Ill.

About five years ago the author encountered an eight-year-old Japanese girl with acatalasemia, *i.e.*, the absence of catalase in the blood. There was a peculiar progressive gangrene starting in the gum of the upper jaw which encroached upon the maxilla, maxillary sinus, and nasal cavity. The infected areas were broadly excised, and on application of hydrogen peroxide during lavage, the wound surface promptly changed into a brownish black color, and no foaming of the hydrogen peroxide solution occurred. Investigations were started to account for this strange phenomenon, and it became apparent that the lack of catalase in the patient's blood was responsible for the blackish discoloration of the blood by hydrogen peroxide. In normal individuals, hydrogen peroxide is decomposed by the catalase in the blood, into $2\,H_2O_2 \longrightarrow 2\,H_2O + O_2$, giving off oxygen in the form of bubbles, and the blood remains a bright red color. In contrast, if there is an absence of catalase in the blood, as in the above mentioned patient, no decomposition of the applied hydrogen peroxide results; therefore, it actively oxidizes the hemoglobin and changes it into methemoglobin, which in turn accounts for the brownish black discoloration. In *in vitro* reaction, upon leaving the reagent for about 30 minutes iron-globin precipitation results leaving a clear supernatant. This supernatant is *propentdyopent*, the final breakdown product of hemoglobin, which shows a positive pentdyopent reaction.

Of the six siblings in this family, four (including the above patient) had acatalasemia. It was common among these four to show a progressive gangrene originating in the teeth. We

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[*] Presented at the 58th Annual Meeting of the American Laryngological, Rhinological, and Otological Society, Inc., Boston, Mass., May, 1954.

[†] From the Department of Otolaryngology, Okayama University Medical School, Okayama, Japan.

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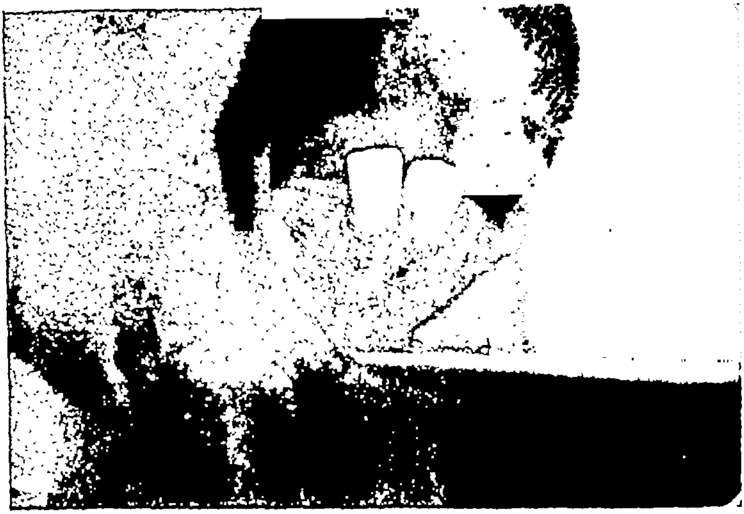
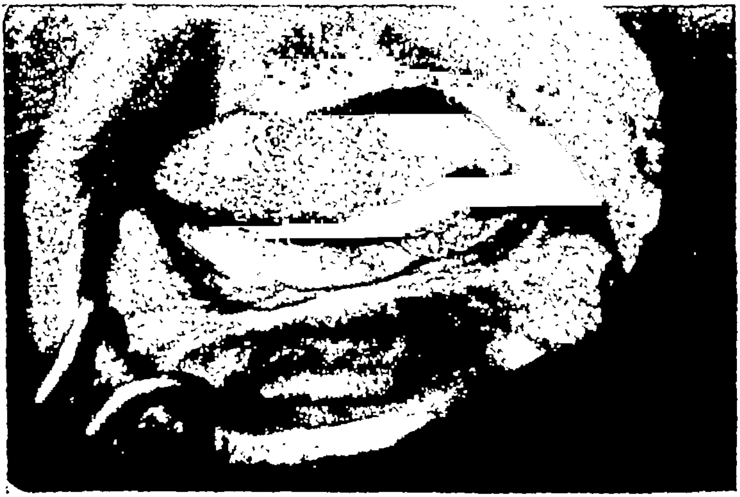


CASE 3. AGE 8, FEMALE, MODERATE TYPE.

Early stadium of ulcer starting around the root of the frontal teeth in the lower jaw.



CASE 5. AGE 16, MALE, SEVERE TYPE.

Though the oral disease is now healed, there can be seen a remarkable recession of the gum. This patient cannot wear denture as the raised area of the gum is too low to be fitted.

CASE 5. AGE 18, MALE, SEVERE TYPE.

The healed condition of the upper jaw. The picture shows a remarkable recession of gum and cicatricial mucous membrane of the cheek which are characteristic of this disease.

Post-operative perforation communicates with nasal cavity and maxillary sinus.

CASE 5. AGE 18, MALE, SEVERE TYPE.

The ulcer which started around the back tooth of lower jaw, is gradually developing into the lateral surface of tongue. Also slight ulcer on the mucous membrane of cheek is noticeable. Most of the tooth had already fallen out.

suspected a relationship between the peculiar dental lesions and acatalasemia, and subsequent studies have verified this impression.

A continual vigilance, in the belief that other cases of similar character existed, was rewarded with two additional families which added five more cases to my series. Thus a total of three family lines with nine cases have now been investigated. Observations during the past four years indicated that catalase value of the blood in all nine cases were invariably zero, with no rise at all when tests were made.

Since my first report, 1947, seven corroborative cases of acatalasemia have been added to my nine cases, making a total of 16 cases in Japan. Fourteen of these 16 cases, namely over 80 per cent, showed the peculiar and characteristic oral gangrene during their childhood, from the age of two to ten. In all, the lesion was what may be termed a serious alveolar pyorrhea or a slowly progressing type of noma, and unless adequate treatment was given to check the progress it would progress and occasionally bring about serious systemic symptoms.

In the history of our nine cases, there are no records of receiving irradiation as from Atomic Bomb or X-ray treatment, and each of 16 cases occurred in several different places in Japan. This suggests that they have no tendency to be characteristic of certain areas.

The treatment consisted of extraction with thorough and extensive excision of the gangrenous bones and involved soft tissues. The results were usually good. Continual rinsing of the infected regions and administration of penicillin also proved an effective adjunct.

It should be added that consanguineous marriages were noted in the parents of all three families. This coupled with the fact that all nine cases of acatalasemia which have come under my observation were present in these families have led me to believe that hereditary factors are undoubtedly responsible or influence this unusual condition. The transmission is believed to be of the Mendelian recessive monogenetic character.

This study was presented in colored motion pictures.

A CASE OF OTITIS EXTERNA APPARENTLY CAUSED BY SALMONELLA FLORIDA.[*]

William Killinger,[†]
Margaret Wynn,[†]
and
Florence Young,[‡]
Jacksonville, Fla.

The literature contains reports of the isolation of Salmonella in a wide variety of clinical entities, including otitis media. We believe this is the first report of Salmonella infection in otitis externa.

The case here reported was one of more than 500 observed during the summer of 1953 in a special study of otitis externa at the U. S. Naval Air Station, Jacksonville, Florida. Cultures were taken routinely from each affected ear on admission of the patient to the clinic and three times a week during clinical follow-up. The specimen was obtained by inserting a sterile cotton swab into the ear canal, then placing the swab at once in thioglycollate broth. In mid-afternoon the cultures were transported to the laboratories of the Florida State Board of Health for examination. When the specimens arrived, ordinarily two to six hours after the culture was taken, plates of eosin methylene-blue and blood agar were inoculated by a standard loop.

The patient, a 21-year-old white enlisted man, was admitted to the clinic on July 8, 1953. He complained of pain in the right ear, unilateral difficulty in hearing, a feeling of fullness in the ear canal, and discomfort on opening the mouth and on biting. The auricle was tender on manipulation. Examination revealed mild erythema and edema of the auditory canal and a scanty gray discharge. The drum was well visualized and found to be normal. A culture was taken in the routine manner. The ear canal was irrigated with 3 per cent saline, and a wick saturated with terramycin-polymyxin B solution, was inserted. The pa-

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[*] Studies of Otitis Externa were conducted under Contract AF 33 (038) 38643 with the USAF School of Aviation Medicine, Randolph Field, Texas, Dept. of Otolaryngology, Washington University School of Medicine, St. Louis, Missouri (B. H. Senturia, M.D., Director), and Contract AF 33 (038)-12466 with the Bureau of Laboratories, Florida State Board of Health, Jacksonville, Florida (A. V. Hardy, M.D., Director).

[†] Medical students at Tulane and Miami Universities respectively, serving clinic assistants during the summer.

[‡] Bacteriologist, Florida State Board of Health.

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tient was also given 1 cc. of the terramycin-polymyxin B solution, in a dropper bottle with instructions to apply two drops into the ear canal every three hours. The culture was submitted for examination in the usual way.

The patient returned the following day according to clinic routine and reported he was much improved and free of discomfort. Examination revealed less erythema and edema and a small amount of exudate in the canal. Treatment was repeated as on the preceding day. He was unable to return for 15 days (July 23, 1953), at which time he reported that he had had no further trouble with his ear and had stopped using the drops at the end of the second day of treatment. His ear canal was normal and a repeat culture revealed only a non-hemolytic *staphylococcus albus*. Two fecal cultures were obtained, and these were negative for enteric pathogens.

The original ear culture, when plated routinely to eosin methylene-blue and blood agar plates, revealed virtually a pure growth of *S. florida*. There were numerous colonies indicating that the purulent exudate inoculated contained large numbers of organisms. All colonies picked gave the typical biochemical and serologic reactions of *S. florida*.* In vitro sensitivity tests indicated that the organism was very sensitive to terramycin and sulfadiazine.

This case is of interest as the first reported *Salmonella* infection of the external auditory canal. Bacteriologic observations have clearly established that Gram-negative organisms, notably Pseudomonas, are commonly encountered in this condition. Although rarely occurring in the normal ear, these organisms are found in large numbers in the infected external auditory canal.

The observation reported here indicates that a known enteric pathogen, also a Gram-negative organism, occasionally may grow luxuriantly in the ear canal and presumably may cause external otitis. The public health implication of this finding is significant, since obviously a food handler with an ear infection discharging an enteric pathogen could contaminate food and be the source of epidemic disease. The prompt clinical response suggested that the terramycin-polymyxin B solution was an effective therapeutic agent. There was no indication as to the probable source of the infecting organism.

To evaluate dependably the frequence of occurrence of Salmonella in otitis externa, more sensitive culture techniques for this organism would be required. The use of an appropriate enrichment, either tetrathionate or selenite F, in an adequate series of cases would be justified.

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* The identification and typing were confirmed by Mildred Galton, Bacteriologist, U. S. Public Health Service, assigned to Florida.

UNUSUAL FOREIGN BODY (PORCUPINE QUILL) IN THE ESOPHAGUS. CASE REPORT.

H. D. HARLOWE, M.D.,

Miles City, Mont.

Foreign bodies of the esophagus are not infrequent or uncommon, but occasionally unusual ones are found. From a review of the literature, Hill[1] reported two cases of porcupine quills as laryngeal foreign bodies, but none have been found in the esophagus. According to Hill,[1] "The ingestion of porcupine quills is extremely rare, and it seems fortunate that they tend to implant themselves firmly in any adjacent tissue and thus remain in an easily accessible region."

Matheson,[2] in reviewing over 600 cases of foreign bodies of the esophagus, stresses the importance of an esophagoscopy even after negative X-ray in any case of suspected foreign body of the esophagus.

REPORT OF CASE.

History: The patient, P. N. E., a white female, age 87 years and edentulous, stated that six days previously she was eating hamburger and believed she swallowed a small sharp piece of bone. She consulted her physician and was told that it was probably just a scratch of her esophagus and to return in a few days if it did not improve. She returned in five days, still complaining of dysphagia and pain in the region of the cricopharyngeus. The patient was referred with a tentative diagnosis of possible foreign body of the cervical esophagus.

Treatment and Course: Preliminary X-rays revealed no definite evidence of any radiopaque foreign body of the esophagus; however, on the lateral cervical esophagus film made after the swallowing of thin barium, a narrow non-opaque foreign body showed in the cervical esophagus outlined by adhering barium at the level of the fifth cervical vertebra.

An esophagoscopy was performed, and what was assumed to be a small sliver of bone was removed with little difficulty. The patient was observed for two days following the operation and then discharged recovered.

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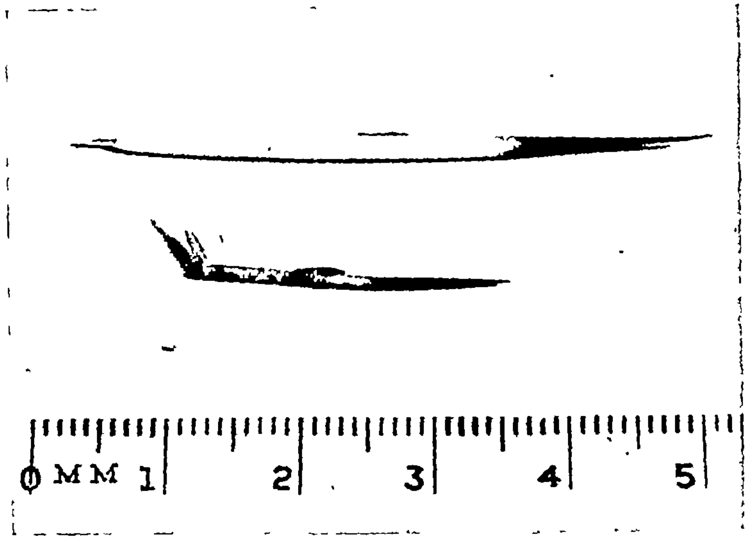
COMMENT.

The foreign body was later found to be a "porcupine quill." The hamburger the patient had eaten was made from deer and pork meat. The porcupine quill had passed through the meat grinder and one portion of the quill shows where it was damaged. The quill measured over 25 mm. in length, and the larger one in the picture is for comparison only.



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Garberson Clinic.

AMERICAN OTORHINOLOGIC SOCIETY FOR PLASTIC SURGERY, INC.

The American Otorhinologic Society for Plastic Surgery, Inc., will hold its meeting at the Waldorf-Astoria Hotel, New York City, September 19, 1954. Participants in the program will be Drs. W. A. McNichols, Sr., J. G. Gilbert, Harry Neivert, Matthew S. Ersner, John J. Conley, and John Jenney. For further information contact Dr. L. J. Feit, 66 Park Ave., New York (16), N. Y.

SUSPENSION FIXATION GEAR POWER LARYNGOSCOPY (WITH MOTION PICTURES).*

ROBERT B. LEWY, M.D.,
Chicago, Ill.

The material which I present is specifically dependent upon considerable prior history and the inventive accomplishments of others. The formulation of both the instruments and instrument combination depends upon work dating back at least 100 years. In direct laryngoscopy, reference should first be made to the work of Desmoreaux in 1853. This was followed in turn by that of Kirstein and shortly later Killian; both contributed to the direct tubular view. Important accessories to the tubular instruments were devised by Seiffert and Haslinger with the former contributing a chest support, and the latter improving somewhat on this with a gear system. Marked improvements were made with a manual distal lighted tube by Jackson.

Jackson's contributions on teaching of proper procedure were of great importance. The open field of suspension work, while started by Killian, received its greatest impetus from R. C. Lynch. For important and extensive cord or tumor surgery this is the method of choice in many clinics.

The instrument combination which I present is a lineal descendant of the laryngoscope of Seiffert and Jackson. The former modified by Haslinger, Roberts, Andrews and Somers. The instrument combination contains some of the strongest and simplest characteristics of each, I trust with the best features in one system. The position method similarly is an easy working simplification.

In my personal experience with the standard method of head holding in the "Boyce position" and with the usual tubular laryngoscope used without mechanical aid, there was often a problem. I found I was occasionally neither strong

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* Presented at the 58th Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Boston, Mass., May 26, 1954.

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enough nor perhaps skillful enough to expose the anterior commissure without great difficulty. There was as well an occasional necessity for general anesthesia or its equivalent. For some years I presumed that this was a personal problem, and let it go at that. Finally, mustering up some courage, I began to inquire among my colleagues. I drew some cold comfort from the fact that many of them candidly admitted that they had frequently contended with similar situations.

A fresh approach to the problem was instituted when one of our residents at the Hines Veterans Hospital made a copy of the window casement opener laryngoscope holder that Lt. Col. Somers presented in the April, 1952, *Archives of Otolaryngology*. This was a home-made imitation of the Andrews detachable laryngoscope holder; however, it possessed a striking difference: there was adequate available power (something Somers did not describe) through the gear system of the casement opener. This was a combined sprocket and worm gear.

Fig. 1. X-ray Laryngoscope with Holder.

There were large gear teeth that moved easily and rapidly. These could not slip, since they integrated a vertical gear system into an almost horizontal groove system of the upright worm.

Using this feature of the Somers instrument as the most essential factor, this power gear was made the functional center of a detachable holder. This had a strong, easily used three-point grip which grasped the laryngoscope handle.

In an independent venture a horizontal bar was put on the Lynch apparatus. With this modification the gallows and the table clamp were dispensed with, and the laryngoscope holder substituted in their place.

We have found that the gear power lifts the tip of the laryngoscope and exposes the anterior commissure gradually and easily. We have used it repeatedly, now in many of scores of cases and thus far with only two failures. Once the necessary exposure is attained, two hands and a relaxed and balanced body position are available for biopsy, removal of polyp or fibroma, papilloma or for exacting examination of the subglottic area after retracting a vocal cord.

Contrary to the orthodox teaching, we found no difficulty in performing laryngoscopy with the neck extended and the vertex of the head on the table as the motion picture will show. This position seemed of itself to hold the shoulders down, making it possible to dispense with either mechanical or human head holders, and thus to reduce by two the team membership requirements of other methods.

The next slide is an X-ray film of a patient thus positioned and with the instrument in place. The mouth and larynx are in a direct line. The neck is extended and the laryngoscope clears the cervical spine easily, as you can see.

In the motion picture that we are about to show you, we will demonstrate a non-standard position for direct laryngoscopy and an available power system attachable to standard laryngoscopes.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

August 1, 1954.

Acousticon Models A-17, A-180 and A-185.
Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 1, New York.

Auditone Models 11 and 15.
Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67 and 70.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac Model M; Mono-Pac Model "Lyric"; Mono-Pac Model "Rhapsody."
Manufacturer: Beltone Hearing Aid Co., 2900 West 36th St., Chicago 32, Ill.

Cleartone Model 700.
Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Junior Model D-2; Dahlberg Model D-3 Tru-Sonic; Dahlberg Model D-4 Tru-Sonic.
Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Fortiphone Models 19-LR; 20A; 21-C and 22.
Manufacturer: Fortiphone Limited, Fortiphone House, 247 Regent St., London W. 1, England.
Distributor: Anton Heilman, 75 Madison Ave., New York 16, N. Y.

Gem Hearing Aid Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.
Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.
Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico Model J; Maico Top Secret Model L; Maico Maxitone.
Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis, Minn.

Micronic Model "Mercury."
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Model T-10; Microtone Model T-612.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

Normatone Model C and Model D-53.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.

Distributor: Normatone Hearing Aid Co., 22 East 7th St., St. Paul (1), Minn.

Otarion Models B-15 and B-30; Otarion Models F-1, and F-3; Otarion Model H-1; Custom "5."

Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40, Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny Myte).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Model 62 Starlet; Model 72; Model 82 (Zephyr).

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Distributor: Radioear Corp., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Silvertone Model H-16, J-92; Silvertone Model P-15.

Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.

Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966; Sonotone Model 977; Sonotone Model 988.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Televox Model E.

Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 99; Telex Model 200; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 953; Telex Model 1700.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.
Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster; Model Cameo.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill

Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal; Zenith "Regent."
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices have vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TRANSISTOR HEARING AIDS ACCEPTED.

Acousticon Model A300; 1 transistor, 2 tubes. Model A-310; 1 transistor; 2 tubes. Model A-330; 3 transistors. Model A-335; 3 transistors.
Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 35, New York.

Audivox, Model 71; 3 transistors.
Manufacturer: Audivox, Inc., 123 Worcester St., Boston 18, Mass.

Beltone Concerto Model; 3 transistors.
Manufacturer: Beltone Hearing Aid Co., 2900 W. 36th St., Chicago 32. Illinois.

Maico Transist-Ear, Model O; 3 transistors.
Manufacturer: The Maico Company, Inc., 21 N. 3rd St., Minneapolis 1, Minnesota.

Micronic "All American" Hearing Aid; 3 transistors.
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Model T1 (Red Dot); 3 transistors. Microtone Model T1 (Yellow Dot); 3 transistors. Microtone Model T31 (Micro-Mite); 1 transistor and 2 tubes.
Manufacturer: The Microtone Corporation, Ford Parkway on the Mississippi, St. Paul 1, Minn.

Otarion Model C-15; 1 transistor, 2 tubes. Otarion Model D-1; 3 transistors. Otarion Model F-22; 1 transistor, 2 tubes.

Manufacturer: Otarion, Inc., 4757 N. Ravenswood Ave., Chicago 40, Ill.

Radioear Model 820; 3 transistors.

Manufacturer: E. A. Myers & Sons, Inc., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Silvertone Model H-25; 3 transistors.

Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.
Distributor: Sears, Roebuck & Co., 925 South Homan Ave., Chicago 7, Illinois.

Sonotone Model 1010; 1 transistor, 2 tubes. Model No. 1111; 3 transistors.

Manufacturer: Sonotone Corporation, Elmsford, N. Y.

Telex Model 954; 1 transistor, 2 tubes. Telex Model 956; 3 transistors.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Unex Model TR-3D; 3 transistors.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Zenith Model Royal-T; 3 transistors. Zenith Model Super Royal-T; 3 transistors.

Manufacturer: Zenith Radio Corp., 5801 W. Dickens Ave., Chicago 39, Illinois.

SEMI PORTABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).

Manufacturer: A. M. Brooks Co., 1222 W. Washington Blvd., Los Angeles 7, Calif.

Aurex Hearing Aids (three types).

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.

Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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THE
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RHINOLOGY IN CHILDREN.

RESUME OF THE LITERATURE FOR 1953.

D. E. S. WISHART, M.D., J. B. WHALEY, M.D.,
and
W. B. WALLACE, M.D.,
Toronto, Canada.

This resumé of the literature on Rhinology in Children comes from the Hospital for Sick Children in Toronto and the Department of Otolaryngology of the University of Toronto. The medical journals, including the pediatric publications of the United States, Britain and Canada have been reviewed. The classification of previous years has been maintained.

GENERAL ARTICLES.

Moncrieff[1] considers that infection in the newborn baby is much more prevalent than is commonly believed. To support this he quotes the 1947 figures of Agnes Macgregor who, working in Edinburgh, found infection to be the cause of neonatal death in 190 out of 618 infants, or 30.7 per cent. It was responsible for two out of every three deaths after the third day of life. He states that sepsis must be recognized if it is to be treated efficiently and that, unfortunately, there is evidence that the condition is often overlooked. To support this he states, *inter alia*, that at one hospital, figures show that in the first three days of life deaths due to infection, proved *post-mortem*, accounted for 15.4 per cent of the total deaths, whereas on the clinical diagnosis only 3.5 per cent were recorded as due to infection.

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication July 26, 1954.

These facts are his arguments for presenting a long and well-planned discussion of the subject from a pediatric point of view.

One part of this discussion is germane to our resumé, for he states that the common cold is usually completely overlooked as a very common cause of the underlying trouble in these infants. When it kills, as it may quite often do in the newborn, and especially in the premature infant, it kills by broncho-pneumonia, or by gastro-enteritis. What seems clear is that it is often missed as a clinical entity. He points out that in a 1949 report by the Ministry of Health, for which he was partly responsible, covering the whole subject of neonatal mortality and morbidity, the section on infection in the newborn fails to mention the common cold.

Why is the common cold not recognized in the newborn? It is because the pattern of disease is different from the pattern in adults and so variable as to give little direct help in diagnosis, which has largely to be on circumstantial evidence.

Consider the clinical features of a small baby with a cold. Lying horizontally in bed, he seldom sneezes, and such excess secretion as is present from the nasal mucous membrane passes down the back of the throat. Obstruction of the nose may occur from time to time, leading in the early days of life to breathing difficulties, often quite wrongly attributed to "asphyxia neonatorum." The obstruction also leads to great difficulty over feeding, because the infant cannot suck properly. The result of this may often be engorgement of the breasts in the mother and failure of lactation — or the baby is weaned because he will not take the breast. The diagnosis of intracranial hemorrhage may be made at this point. Sometimes, but in his experience, rarely, some degree of snuffles and anterior canal discharge occur, often leading to a suspicion of syphilis and delay while this is excluded. By now the infant may have begun to vomit or have green stools as a manifestation of his parenteral infection.

Alternatively, infection may have spread down the respiratory tract and signs of involvement of the finer bronchi and alveoli of the lungs appear. Whether or not the primary infection of the nose was a virus, there is by now secondary invasion — staphylococcal, or one of the Gram-negative group. All this may happen with surprising rapidity, and death may occur within a few days of the onset, again without any suspicion that the original infection was essentially of the upper respiratory tract. Again and again with this picture it is possible to track the infection back to the baby's contact with an adult who has a nasal infection. The mother herself may have a chronic sinus infection (as in one of his patients), or the trouble may have come from one of the nursing staff (in one instance a night nurse in a nursing-home where at that period the matron did not "hold" with masks). Things are perhaps a little better than they were, because the use of masks is more widespread, but so long as the cold is largely unrecognized in the baby for what it is, the menace will continue.

Diagnosis must precede treatment and if possible should include identification of the infecting microorganisms. This will put the use of chemotherapy and antibiotics on a rational basis. Breast feeding must be continued. Dehydration must be dealt with on the usual basis. The hemoglobin level must be watched.

This is a very brief summary of what may be a complicated and prolonged campaign of treatment. More important is the *prevention* of infection. A high standard of personal hygiene must be scrupulously observed by all concerned. The use of efficient masks by the nurses and visiting staff is, he thinks, all important, although there is a tendency to decry their value.

Rhoads[2] has written an excellent paper concerning the joint responsibility of the otolaryngologist and the internist in the removal of focal of infection.

He points out that there has been a waning of enthusiasm for removal of all accessible tissue suspected of harboring pathogenic bacteria. Some of the reasons have been: *1.* Failure of patients with systemic disease to get well in a number of instances after removal of foci of infection; *2.* The observation that many people with obvious active foci of infection do not suffer from systemic maladies usually attributed to such foci; *3.* Failure to demonstrate any focus of infection in many patients with such diseases as long standing rheumatoid arthritis, subacute bacterial endocarditis and nephritis; *4.* Finally the failure to completely remove foci of infection so that it is possible to demonstrate tonsil remnants in 72 per cent of young adults who have previously undergone tonsillectomy.

Rhoads feels that the excesses of the first two decades of the century are to be deplored but points out that the above arguments against the importance of focal infection do not hold water.

He states that no one can deny that bacteria growing in thrombi at the roots of infected teeth or in tonsillar crypts are frequently the cause of subacute bacterial endocarditis. Also who can deny that bacteria flourishing in a localized focus by their toxins cause remote effects such as the rash of scarlet fever and the paralysis and myocardial lesions of diphtheria.

Modern medical literature is now suggesting that an important effect of low grade infection is the sensitization of certain tissues of the body so that they hyper-react to super imposed infection giving rise to the so-called collagen diseases, such as rheumatic fever, disseminated lupus erythematosis and probably acute nephritis.

Why some humans can harbor continuing low grade infections over long periods without developing systemic disease is poorly understood; but it does not invalidate the possibility that infection in others may act as so-called tissue sensitizers, and failure to observe swift improvement in systemic disease following removal of foci does not prove that the focus was not involved in the entire phenomenon. He cites a parallel that plugging the hole in a boat to remove water already accumulated will not be effective, but it does not prove that the hole was not responsible for the accumulation of the water.

Careful examination and bacteriological studies may be necessary to prove a focus of infection does exist. A table showing the organisms normally found in nose and throat, and those organisms which can be considered as pathogens accompanies the paper.

Throat cultures may be misleading because frequently ordinary throat cultures may reveal no pathogens whereas cultures made by grinding excised tonsils from the same patient in a mortar with sand and broth will lead to recovery of streptococci.

Since the advent of sulfa and the antibiotics even deep seated infections can be cleared up by vigorous drug therapy, but there remains a considerable proportion where the foci persist despite conservative treatment and prevention of such chronicity is the joint responsibility of the internist and the otolaryngologist.

Tonsillectomy and adenoidectomy, surgical drainage of sinuses, incision of ear drums and frequently mastoidectomy will continue to be necessary to prevent at least in part, the chronic systemic disorders such as rheumatic fever, rheumatoid arthritis, iritis, bronchiectasis, chronic anemia and nephritis.

The physician is almost daily confronted with evidence of the impact of weather and climate on ear, nose and throat diseases.

Kinkade[3] discusses this topic and points out that the entire human organism is immersed in an ocean of air which affects every physiologic function.

There are constant changes taking place in the surrounding air to which the organism must adjust satisfactorily. Some people make these physiological responses readily, while others may react so massively that in the upper respiratory system complete blockage of the nose occurs with much sneezing and profuse mucoid discharge.

These individuals are said to suffer from vasomotor rhinitis due to domination by the sympathetic nervous system.

Sudden chilling, drafts, air pollution, rapid changes in temperature, and pressure fronts which lead to climatic crises are all factors which require adjustments on the part of the exposed individual. Failure to respond satisfactorily can lead to sinus infection, sore throat, Eustachian tubal catarrh or perhaps epistaxis such as in mountain sickness due to thinning of air.

Humans vary in their reactions to these stimuli, *e.g.* allergic subjects and those with vasomotor rhinitis tend to over respond. The result is that frequently the question arises as to the advisability of recommending a change of climate for a patient. The decision is not easy, and at the present state of knowledge the physiology of climatic response does not lend itself to comprehensive generalization; but with careful selection of cases, climatic therapy is a highly flexible adjunct to otolaryngology. Additional observations and continued research may be expected to improve our understanding of the very definite effects of weather and climate on the upper respiratory tract.

In discussing the thymus problem Littleton, Motsay and Perry[4] were unable to show any compression of the trachea by the thymus gland. They used X-rays, fluoroscopic examination and cadaver experiments with iodized oil (Lipiodol). They disagree with many autopsy descriptions of sudden death being caused by tracheal compression by the thymus gland. They have had no experience with status thymico-lymphaticus and agree with former writers that there is no evidence that status thymico-lymphaticus exists as a pathological entity. They do believe that Roentgen therapy has a beneficial effect on low grade chronic or recurrent tracheobronchitis which may produce stridor and cough and sometimes cyanosis.

SINUSITIS.

Dingley[5] states that the aim of all nasal surgery should be to achieve the end result with as little trauma to intranasal structures and sinus mucosa and as little interference with nasal physiology as is consistent with success. He then discusses generally temporary drainage and permanent drainage of the maxillary antrum, and finally contrasts intranasal antrostomy and the Caldwell-Luc operation under the following headings: accessibility of the surgical approach, surgical technique and instruments employed, indications for each operation and the disadvantages of each method. His arguments will repay study.

Most of his paper deals with the adult, but he has this to say regarding children.

"In children up to the age of 10 years permanent drainage is well-nigh impossible to attain and bone if traumatized will make every endeavor to regenerate. The antro-nasal wall is sometimes surprisingly thick and tough; consequently, operations for drainage are usually unsuccessful, and if attempted are likely to result in closure or contracture to a minute opening useless for drainage purposes. The consequent trauma to intranasal tissues, and possible damage to tooth buds appears unjustifiable, since so little is gained at such expense. Children react better than adults to puncture and wash-out, but seem to resent trauma to their intranasal mechanism even more than the latter. Polythene tubes are most useful.

A paper on the incidence of chronic infection of the maxillary sinus in children has been written by Mawson and Gray.[6] In 50 children admitted for routine removal of tonsils and adenoids they found an incidence of unsuspected pathological infection of the antra of 22 per cent. In another series of children who were suspected to have antral infections the washings were found to be infected in 45.4 per cent.

In these investigations the washings were obtained by a two cannula method and the specimen was centrifuged for 10 minutes. After the supernatant fluid was removed the concentrate was examined by direct smear and was cultured as well. With increasing experience the films were found to be more informative than the cultures.

The examination of the direct smears on films suggested that they could be grouped into four categories:

I. Antral response to trauma. These films contained a large number of red cells, a few well stained polymorphs and isolated well stained epithelial cells in which the distinction between the nucleus and cytoplasm was well defined. There was no fibrin. No cellular debris and no organisms were seen.

II. Short standing antral infections. A little fibrin was present and a variable number of well stained polymorphs Monocytes were seen and scattered well stained epithelial cells together with the occasional smudge cells. Organisms were present in variable numbers.

III. Early chronic antral infections. A moderate amount of fibrin. The polymorphs have lost the staining differentiation between nucleus and cytoplasm and there are many smudge cells. There was much debris and organisms were present in variable numbers.

IV. Late chronic antral infections. These films showed much fibrin and all characteristics were difficult to make out because most of them had smudged. Epithelial masses were present but resembled lumps of amorphous debris. Organisms present stained in such a way as to suggest they were dead. Gradual cellular lysis had continued so that the end result was an acellular network of fibrin containing a few scattered organisms.

With the exception of the traumatic group these categories were not well demarcated and tended to merge into one another.

This is a very interesting paper and contains a section dealing with literature on this subject and discussion of controversial points.

Davison[7] discusses hyperplastic sinusitis and the results of his treatment of 50 cases. He feels that the polypoid thickening of the nasal sinus mucosa is best explained on the basis of local sensitization of the submucosal tissues to bacterial proteins.

These patients usually can recall that their symptoms began with an acute severe febrile respiratory infection. Strangely enough they seldom complain of headache.

After careful history taking, in addition to the usual ear, nose and throat examination, he takes smears and cultures, X-rays the sinuses, and lavages all cloudy antra as a diagnostic procedure.

Because these patients may be sensitive to other than bacterial proteins, a careful history in that respect is important, and skin tests should be done when indicated.

Microscopically the tissue changes are excessive edema, increased numbers of eosinophils and relatively fewer neutrophils. These appearances give the impression that they are reversible changes, and he has felt that the best method of treatment was to use large bacteriocidal doses of penicillin plus cortisone to shrink the edematous tissue. Surgical procedures are reduced to a minimum.

Best results have been obtained by using doses like two million units of fortified penicillin every four hours for a period of at least a week along with Cortisone, which is kept up for two weeks, starting with an initial daily dose of 200 mgms., but reducing this so that the total dose averaged about 900 mgm. per patient.

Skolnik, *et al.*[4] have reported on rhinogenous complications in the antibiotic era. Widespread use of these drugs has not eradicated severe intracranial complications of acute and chronic infections of the nose and paranasal sinuses.

The neurological manifestations do not always conform to a classical pattern, but the otolaryngologist must be alert to recognize the neurological symptoms and signs and must be able to treat the primary focus and the complications.

In all types of intracranial infections which arise by spread from the nose or sinuses four tissues are exposed — nasal and sinus mucosa, bone, meninges and brain. The spread may be by direct extension or contiguity, or it may be by continuity along the blood vessels through bony dehiscences or along the sheaths of the olfactory nerves.

The following complications can occur: *1.* Extradural abcess occurs more commonly by direct extension backwards from an infected frontal sinus, usually with associated osteomyelitic changes. *2.* Subdural abscess or subdural empyema is due to inflammation of the inner surface of the dura, which usually produces a purulent exudate. If the spread is by direct contiguity, it is easily recognizable at operation, but in some cases infection may travel by way of anastomosing veins from sinus mucosa to inner layer of dura without producing inflammation of the outer layer. In the latter patients the relatively normal appearance of the dura when exposed at operation must not be permitted to prevent adequate search if the symptoms warrant it. *3.* Brain abscess. Again extension may be by contiguity or by continuity. If by contiguity, gross changes occur in the bone and dura, but when spread is by continuity brain abscess may develop with no significant changes in the overlying bone and dura. Brunner found normal bone in 70 per cent of brain abscesses, and normal external dura in 44 per cent.

The authors then present and discuss five cases, and the information as to diagnoses and management is well worth reading in its entirety.

Lillie[9] points out that the occasional intranasal cyst may rupture and discharge varying amounts of fluid. This fluid contains both chlorides and sugar, and may easily be misinterpreted as being cerebrospinal fluid, unless the physician is aware that fluid from intranasal cysts can contain moderate amounts of chloride and sugar.

The chief distinguishing features are that fluid from the intranasal cysts is yellow as compared to clear spinal fluid, also that fluid from intranasal cysts will clot readily if allowed to stand.

Ornston[10] reports on the new polyethylene spray bottles which are now being marketed as a means of administering many well known nasal medications.

He evaluated the bottle in three ways. First by observing the nasal coverage of sufathiazole crystals obtained by its use. Second by comparing the results of spray and drop medication in 49 patients and lastly by making X-ray studies of a radio opaque medium administered by drop and spray bottle method.

He found that the effectiveness of the spray depended upon the potency of the common meatus. If two applications are used the first spraying will open the meatus enough for the second spraying to reach the olfactory fissure.

In the clinical study on 49 patients there was no important observable difference in the effectiveness of the two methods. The spray bottle seemed to provide adequate coverage.

X-ray examinations showed that coverage by properly applied drops was superior — but that coverage by spray bottle was effective, and providing two sprayings are used, one shortly after the other, the spray bottle method is probably the treatment of choice for those patients who find it impossible or inconvenient to use the drops properly.

ALLERGY.

James, Brand and Pepys[11] emphasized the growing respect for the allergic aspect in the treatment of children having upper respiratory infection and the need for a concerted approach by the otorhinolaryngologist, the pediatrician and the allergist. All the speakers agreed that the diagnosis of allergy is based on the clinical history, a personal or family history of allergice disorders and the presence of eosinophil cells in the nasal smear.

The smooth swelling of the sinus mucosa seen on radiography and its rapid reversibility were described; also the rubbing and flattening of the nose, aptly termed the "allergic salute" by which these patients try to relieve the typical and often intense itch of allergic conditions. While it was shown there was a case for "bacterial allergy" it was also pointed out bacterial allergy was not universally accepted as a cause of typical allergic rhinitis. Treatment of allergy before and after any operative treatment was advised.

Allergic rhinitis and paranasal sinusitis in children frequently present the otolaryngologist with a distressing and baffling problem. A new approach to the problem is presented by Harper[12] whose thesis must be studied in its entirety.

He presents the hypothesis that in times of stress "morbid echoes" of obsolete defense reflexes may be manifested which are parallel to, if not co-existent with those anatomical and histological vestiges which link man with long-forgotten ancestors. A convenient example of a reflex defense mechanism of the type under consideration is provided by olfaction. During long eras olfaction was vital to the continuation of the race. Olfaction became supplanted in the recent evolutionary past only when the development of other senses led to its relegation to the very minor role seen in man. It would be remarkable, in his view, if we did not find today manifest in times of stress physiological vestiges of this defense reflex.

In his paper certain clinical aspects of a small representative group of asthmatic and allergic children living at a family school under conditions especially favorable for observation are outlined. A summary of the clinical records of each child mentioned, often covering a period of years, is available on application to the Editor of the B.M.J. or the author.

During recent years there has been a small proportion of severely allergic children at this family school who, previous to arrival, had had much treatment without success. Such children arrive at the school with some or, in certain cases, all of the apparatus and regimen so familiar to everyone who has had to deal with this most difficult and frustrating type of illness. Special bedding, sprays, nose drops, ear drops for the not infrequently associated otitis media, are prominent, together with a compilation of the causes, climatic and dietetic, that have produced attacks. Often copies of prescriptions from medical authorities on allergy and ampoules for frequent injection accompany the child.

For several years it has become the practice at this school to cease every drug and prohibition on arrival, and it is found that the child settles down easily, and soon loses the asthmatic or other allergic manifestations, however severe or protracted the condition may have been. It would seem that the threshold at which an attack can be produced has been raised to a significant degree. The turgescence of the olfactory mucous membrane found in this catarrhal type of child rapidly subsides, the obstructive threat to adequate sinus drainage is removed and the paranasal sinus infections that seem to be an almost constant accompaniment of the asthmatic diathesis quickly clear up. As a result, the existing ear discharges cease and there is a noticeable increase in well-being that is apparent to everyone, and most of all to the child itself. Self-confidence rapidly increases as the child finds that things can be eaten, and done, without producing any attack. Colds which in the past have so frequently produced asthma, often complicated by acute sinus infection and bronchitis, are now only colds and run a normal course.

Now, it is his experience that in this little community the diverse manifestations of allergic disease are unified in that the entire syndrome with its complications improves dramatically, and the rare attacks that occur while the child is within its influence can usually be traced to some threat to security, such as the mishandling or anxious treatment of the children by a member of the staff during the temporary absence of the principal. Another fertile cause of relapse is the arrival of one of the parents or relatives.

Only slight quotations from this arresting paper have and can be given here.

He states the above are examples only of a consistent pattern observed by him in the group of allergic children that have passed through this community. Without drugs, without nursing, without doctoring for their allergy their problems have largely ceased to exist so long as there has been no introduction from without, or from within, of stress, anxiety or insecurity.

Violent reaction to stress is frequently seen in children who are sheltered to an almost abnormal degree form the real danger and climatic changes. How often the intelligent mother of a small family bewails to the doctor as she tends her stressed child: "It seems that the more care I take the more illness she produces; the neighbors' children run the streets without supervision or care, and they seem hardly ever to be ill."

Stewart and Kawa[13] report personal observations and investigations in the therapeutic effects of cortisone and ACTH in 33 cases of nasal allergy, which do not, however, include hay-fever.

The clinical material for this investigation consisted of 33 patients of whom 24 were females and 9 males and their ages ranged from 11 to 61 years. Twelve cases gave a family history of allergy. The diagnosis was based on three main symptoms which generally were present in the following sequence; attacks of sneezing which took place first thing in the morning, followed by nasal obstruction, and then a clear profuse discharge which occurred after the relief of the obstruction. The diagnosis also depended upon the appearance of the interior of the nose as seen by anterior and posterior rhinoscopy; this showed the unmistakable picture of allergy of the soft tissues. An X-ray examination of the paranasal sinuses was also carried out to eliminate any gross underlying infective process. Twelve of the cases showed soft tissue swelling in one or both of the antra, while the remaining 21 cases were normal. In those cases which showed changes, a further X-ray was taken on the conclusion of the initial treatment and all except one showed an improvement in the one containing space and a shrinkage of the swollen soft tissues.

On admission a small piece of tissue from the nasal mucosa was taken for biopsy. The nose was examined each day. Prior to discharge a second biopsy from the same region of the nose as the first was taken. The day following their discharge from the hospital they returned to commence their maintenance treatment as out-patients.

The Cortisone used in the investigations was that manufactured by Merck and Co., of New York. It is in the form of a saline suspension of cortisone acetate.

Cortisone is most commonly given by the intramuscular route by a single daily injection, or, in their experiment, it was better divided and administered at six-hourly intervals during the active stage of treatment. It may also be administered by the mouth either as tablets or in suspension. The tablet is usually 25 mg. or, if in suspension, a similar dose in 4 cc. bulk. The oral route is considered quite satisfactory, but larger doses have been found necessary to achieve the same results as by intramuscular injection.

They employed the ordinary ACTH as supplied by Armour. They gave it intramuscularly in divided doses at six-hourly intervals. They also used it as a nose spray following the initial treatment by the intramuscular route in eight cases. Only in three cases was this method of treatment by itself successful; the other five cases required maintenance doses by injection as well.

They used Cortisone by itself in six cases, Cortisone combined with ACTH in four, and ACTH alone in 23 cases — these groups are designated A, B, and C.

The investigation was undertaken in the past six months, so they have only short-term results of treatment. They must wait for a further period for long-term results.

In group A, three were completely free of symptoms, two much improved and one improved. In group B, two cases were completely relieved, one was much improved, and one failed to continue treatment although he had greatly improved while in hospital. In group C, 14 were reported as being free of symptoms, seven were much improved, one improved and one unimproved.

The authors claim to have evolved a form of treatment for simple and chronic allergic rhinitis where a large proportion of the cases have been free of symptoms or vastly improved.

They consider the treatment reasonably safe if carried out with intelligence and care and if used in selected cases and not indiscriminately.

Shahinian[14] in a paper on vasomotor rhinitis describes this phenomenon as a nasal manifestation of disturbed neurovascular physiology. This disturbance may be due to: *1.* Chronic nasal allergy, *2.* Endocrine or metabolic dysfunction, *3.* Psychomatic personality states, *4.* Miscellaneous factors, such as climatic or thermic conditions and derangements of calcium and starch metabolism.

The ideal treatment, of course, would be to eliminate whichever cause or causes may be giving rise to the disturbance in neurovascular physiology. Often, however, the cause can not be determined, or if determined cannot be eliminated; therefore, the rhinologist must frequently have to resort to some form of nonspecific local therapy to provide relief. There has been a host of local treatments advocated, but the one that appears to offer the most advantages is the method of submucosal galvanocautery.

Of the several outstanding symptoms of vasomotor rhinitis, that of impaired breathing is the most responsive to this method of treatment. Large tumescent turbinates which still will respond to vasoconstriction are the most suitable with less dramatic results if secondary fibrous hypertrophic changes are present.

Shahinian describes his indications and methods of treatment in detail and as well discusses a modified instrument and instructions on its calibration by the rhinologist before use. His results have been most gratifying in 412 patients treated over the past 12 years. Relief of stuffiness may last from two to ten years.

This is a most interesting paper and should be read in full.

ANTIBIOTICS.

Dingley[15] considers there is no field of surgery where warning against the unwise use of antibiotics is more needed than in otolaryngology. It is a basic surgical principle that pus, wherever accessible, requires drainage, and the tendency in some quarters today to substitute these agents in place of surgery should be strongly resisted; thus therapy can be used very early after the onset of acute otitis media with safety and benefit, but it must be accompanied by repeated examinations of ear-drum and hearing in order to see that resolution is taking place.

One aspect of the action of antibiotics which he thinks is not sufficiently realized is their ability to forestall infection of certain organs and tissues. It is impossible to say how many ears and nasal sinuses have escaped infection, particularly in the acute specific fevers, as a result of their attack on the nasopharyngeal infection, but it is certainly a very large number. By their beneficial effect on the blood-stream infection they must frequently prevent pulmonary and renal involvement. They thus provide a prophylaxis against extension of infection hitherto unknown. By doing so much for the patient, however, they induce him to become lazy in his own interests, with the result that efficient immunity is not built up as it would be if the infection had been fought unaided; this may explain the frequent recurrences, often over a short interval, noted in acute otitis media, suggesting that complete recovery from the first attack had never taken place.

Schenck[14] reports on some of the problems encountered in the use of antibiotics in otolaryngology. Entirely new criteria for diagnosis and treatment must be evolved when the otolaryngologist first sees a patient who has received antibiotic therapy for days or weeks or even months.

The problem of preventing hearing loss following acute otitis media has become more complex since the introduction of antibiotic agents. This is because of the erroneous idea that paracentesis has become obsolete: a view held by many general practitioners and pediatricians. Early adequate antibiotic treatment will usually make paracentesis unnecessary, but improperly directed or late therapy will often reduce pain and fever and lead to a false sense of security which may result in unfortunate sequela such as deafness, mastoiditis or even intracranial spread. When frank bulging of the ear drum persists for more than 24 hours in spite of antibiotic therapy, paracentesis is imperative if serious damage to the conduction apparatus is to be avoided. In younger children hearing defects are especially serious and may go undetected for a very long time during a period when adequate hearing is so important to normal progress.

These limitations must be emphasized by the otologist, and hearing tests should be insisted upon after recovery from attacks of otitis media.

New and unusual infections have appeared as another result of antibiotic therapy. There has been an increase in the number of proteus and pseudomonas infections and of moniliasis following the use of wide-spectrum antibiotics. Even such rare causes of human disease as E. coli and aspergillus have appeared as predominating organisms in infections of the upper respiratory tract. Penicillin-resistant staphylococci have become a real danger.

Antibiotic reactions such as glossitis and black or hairy tongue are not uncommon. These lesions may be sensitivity reactions or may be the result of antibiotic induced vitamin B deficiency.

Structural damage may also occur as direct toxic effect of antibiotics. Sometimes loss of taste or smell may result, or loss of vestibular and labyrinthine function from toxic effects of streptomycin or dihydrostreptomycin therapy. Eighth Nerve complications vary directly with the daily dose and the number of days such daily doses of streptomycin or dihydrostreptomycin are administered. They are always bilateral and appear more abruptly with the dihydro-streptomycin and more gradually when streptomycin is used. Slight changes are reversible, but severe damage is permanent. When deafness occurs it is preceded by tinnitus. Dihydrostreptomycin impairs the cochlear mechanism more readily than streptomycin but is less injurious to the vestibular apparatus.

Schenck states 30 to 40 per cent of individuals receiving dihydrostreptomycin acquire a perceptive deafness of 30 or more decibels. The symptoms have appeared after therapy has been discontinued for as long as 24 days. The question has arisen that antibiotics may be a potential cause of prenatal acquired deafness. The foetal auditory apparatus is especially vulnerable during the first three months of gestation but the effects of streptomycin therapy during this period are still unknown.

Bunn[17] points out that there are now available many antibacterial agents which can be of aid to the host in defending himself against invading microorganisms.

Most human infections can be controlled with one or more drugs administered wisely.

He feels that with a few exceptions it is wiser to use one drug alone and in adequate dosage than to use combinations of drugs. He also favors parenteral administration of antibacterial drugs unless the area of infection is so avascular that blood-borne drug is unlikely to reach the lesion in sufficient concentration. Under these circumstances local therapy is advisable.

Duration of therapy should be at least five days and he reports, that following streptococcic sore throat, when antibacterial therapy is stopped in 48 to 72 hours because the local inflammation has subsided, there is a significant increase in the incidence of rheumatic fever and glomerulonephritis.

In meningococcic and pneumococcic meningitis he favors massive parenteral therapy with penicillin and recommends 1,000,000 units q.2h. continued for from 10 — 14 days. This dosage can reduce the mortality rate of otogenic meningitis to less than 25 per cent.

Tuberculosis is an exception which must be treated with combined drugs. Streptomycin with paraaminosalicylic acid or with Isoniazid. These combinations discourage emergence of resistant organisms and reduce the toxicity of the streptomycin. In the latter regard dihydro-streptomycin used over long periods is much more toxic to the eighth cranial nerve than is the parent substance.

He does not feel that antibacterial agents alter the course of or prevent secondary infections if used in treatment of the common cold.

Huang and High[18] compared the results obtained in treating common infections encountered in infants and children with penicillin administered orally every four hours with those obtained when oral penicillin was given every 12 hours. The series included 292 children observed through 308 infections. The conditions treated were all otolaryngological and other upper respiratory infections. Three therapeutic programs using orally administered penicillin were compared. In the first, 200,000 units of potassium penicillin-G in a liquid vehicle were administered as an initial dose, followed by 100,000 units at intervals of four hours. In the second program 300,000 units of the same potassium penicillin-G preparation was given at intervals of 12 hours. The third group consisted of the oral administration, of 300,000 units of procaine penicillin at 12-hour intervals.

The authors found that the patients who received 300,000 units of penicillin at intervals of 12 hours responded as satisfactorily as did those who received 200,000 units as the initial dose, followed by 100,000 units every four hours. The effectiveness of procaine penicillin was comparable to that of potassium penicillin-G when each was administered in equivalent amounts at intervals of 12 hours.

A study of the comparative effect of penicillin, aureomycin and terramycin on streptococcal tonsillitis and pharyngitis was carried out at Warren Airforce Base by Denny et al.[19] A homogenous group of early streptococcal infections was selected by the following criteria; *1.* Exudate on tonsils or pharynx. *2.* A total leukocyte count of 10,000 or greater. *3.* Duration of symptoms less than 31 hours.

Fifty-three patients were treated with penicillin, 53 with aureomycin, 51 with terramycin and 50 received placebos. A beneficial effect was demonstrated in all patients receiving penicillin, aureomycin or terramycin. No one drug was consistently more effective than the other two. The incidence of anorexia, nausea and vomiting was high in the aureomycin treated group, and both aureomycin and terramycin increased the incidence of loose stools. From the standpoint of toxicity, penicillin is the drug of choice.

Penicillin was more effective in the eradication of streptococci from the throat.

The dosage used was: — crystalline procaine penicillin-G., 600,000 units daily for 5 days.

Crystalline aureomycin hydrochloride 1 gm. on admission and 0.5 gm. every 6 hours for 19 additional doses.

Crystalline terramycin hydrochloride 1 gm. on admission and 0.5 gm. every 6 hours for 19 additional doses.

Controls: — lactose placebos every 6 hours for 20 doses.

The absorption of chloramphenicol had been studied in the adult and the child, but there was no adequate information on the newborn. This led O'Brien[20] to investigate the newborn as there were good reasons for supposing that the absorption of the drug may be difficult in the first 10 days of life.

The investigation was based on 178 observations on 68 infants. The palmitate suspension was given just before the feed either by spoon or in the bottle diluted with an equal amount of breast milk or dried milk mixture. The crystalline preparation was individually dispensed and added to the feed as a powder. No difficulty was encountered in giving the drug in either form; however, a number of infants receiving higher doses of both preparations vomited after 24 - 48 hours and continued until the drug was stopped.

Venous samples were taken and assays performed in the laboratory. The results showed that crystalline chloramphenicol was more quickly and completely absorbed in the newborn than the palmitate form, the use of the latter offering no particular advantage. The dosage suggested was 25 mgm. per kg. of the crystalline preparation every six hours. If the palmitate preparation were used the dosage recommended was 100 mg. per kg. initially followed by 25 mg. per kg. every six hours.

TONSILS AND ADENOIDS.

Tremble[21] is of the opinion that the arrangement of lymphoid tissue commonly called the "ring of Waldeyer" is not at all well understood even at the present time. We do know, however, that the adenoid tends to atrophy after puberty and as a rule disappears entirely between 18 and 20 years of age. In view of this it would appear that the various groups of lymphoid tissue act as a means of defense at the entrance of the respiratory and digestive systems in the first ten or 12 years of life.

It should be kept in mind that the majority of children build up a natural immunity to upper respiratory infections between six and seven years of age and this is the period when tonsillectomy is most frequently performed.

When are tonsils normal and when are they abnormal? This is of course a very difficult question to answer. Small chronically infected buried tonsils often show a higher bacterial count than hypertrophied ones containing large crypts. Epithelial debris or cheesy plugs which can often be expressed from the crypts of healthy individuals are not as a rule harmful. On the other hand when thick creamy pus can be pressed out repeatedly, especially in the presence of fibrosis, there is more likelihood of absorption. Every laryngologist knows that many persons harbor hemolytic streptococci in pure culture without any untoward symptoms.

The author thoughtfully covers many aspects of this controversial question and concludes that each case must be decided on its own merits. A careful history in conjunction with thorough examination and long clinical experience are still of the utmost importance.

Proud[22] writes that in a certain metropolitan community in 1951 a hospitalization insurance company paid claims to 1,264 obstetrical cases as against 1,311 claims for tonsillectomy during the same period. This leads to the realization that the operation is still performed with amazing frequency. With the advent of antimicrobial therapy operations for the removal of lymphoid tissue have diminished in number yet certain specific indications remain. Among these are perhaps most commonly mouth-breathing due to enlarged adenoids, and in these cases he recommends pre-operative palpation of the post-nasal space which although distasteful is less so than an unsatisfactory surgical result.

Recurrent attacks of otitis media may cease after adenoidectomy especially if lateral band dissection and removal of the tubal bud are carried out. In this regard he is of the opinion that despite the number of palate retractors and "round-the-corner" devices adenoidectomy is still essentially a blind procedure. Failure of the procedure to achieve results is, therefore, surprisingly rare but does occur if the operation is incompletely done and in cases of irreversible middle ear disease or in autonomic dysfunction of the middle ear membrane.

In cases of persistent secretory otitis not due to autonomic dysfunction, adenoidectomy may bring relief, and he advocates myringotomy at the same time for added assurance of relief.

In case of vasomotor rhinitis further aggravated by adenoid hypertrophy adenoidectomy may achieve gratifying results, but the operation should only be advised after repeated examinations have shown that the adenoid is definitely contributing to the obstruction.

Today adenoidectomy is frequently done without tonsillectomy and these children have a minimum of discomfort and few postoperative complications.

Tonsillar hypertrophy, unless very great and sufficient to cause dysphagia or respiratory distress, is not an indication for removal.

Quinsy and recurrent Vincent's infection are positive indications as well as removal to free an individual of the carrier state of diphtheria.

One or two mild attacks of tonsillitis are no indication for surgery, but frequent or unusually severe attacks are indications for operation.

The ideal age for tonsillectomy is when the trouble begins.

Blassingame[23] pleads for more realism in the surgery of the tonsils and adenoids.

At least 50 per cent of the cases are operated upon by individuals unqualified by training to perform this operation.

Because of the importance of pharyngeal lymphoid tissue in infections of this area, thorough removal is necessary. Complete extirpation should include the faucial tonsils, the lingual tonsil, the pharyngeal tonsil, plus all secondary lymph nodes.

The faucial tonsil offers the least difficulty, but unfortunately the importance of preserving the posterior pillars is not fully realized. The elimination of one or both pillars distorts the palate and may affect the modulation of the voice.

The lingual tonsil warrants removal in at least 85 per cent of tonsillectomized patients and must be done carefully.

He urges most thorough removal of pharyngeal tonsil and advocates the Yankauer nasopharyngeal speculum and an assortment of punches for removal of lymphoid tissue in the lateral pharyngeal recess.

Unger[24] has devised an instrument which he has found reduces immediate operative hemorrhage after removal of adenoids and also lessens the possibility of secondary bleeding.

He feels that both primary and secondary bleeding are more frequent from the adenoid site than from the tonsil beds, because the adenoids are removed with sharp instruments, and, therefore, the area is deprived of the tearing and crushing of the tissues that occurs during tonsillectomy.

His instrument is a knurled roller fitted to the handle and shaft of an instrument that is much like an ordinary adenoid curette. As soon as the adenoid has been removed, the area is rolled with strong pressure to compress and lacerate the bleeding tissues, so that the openings of the blood vessels are crushed together and closed. The rolling also serves to crush any small flat collections of adenoid tissue that may have escaped the blade of the adenotome

McKenzie[23] reviews the risks of tonsillectomy. He gives an account of a number of disasters which were the experience of one surgeon, although some of them were under the care of colleagues; but each disaster was so unusual and so tragic that it was recounted in detail and discussed by the whole hospital staff.

The risks of today, he admits, are fewer than those of 20 years ago. The chance of serious infection has nearly disappeared. Acute otitis media and its sequelae are much less frequent. The anesthetic risk is less now when the anesthetist is of specialist rank.

He believes that for tonsillectomy the surgeon needs a patient *who is deeply anesthetized and has had a smooth induction* of anethesia. He finds that the inexpert anesthetist keeps his patient under too light anesthesia. The best anesthetic he has seen for tonsillectomy is intravenous thiopentone combined with an intratracheal tube which delivers oxygen.

He divides the risks into four groups: those at the time of operation, those immediately after operation, those within 24 hours, and those later than 24 hours after operation.

At operation the risk that is uppermost in the mind of the surgeon is hemorrhage. He states that it is unfortunately true that the neater the surgical work the greater is the risk of bleeding at the time of operation.

During recovery from the anesthetic the patient should be kept on his side where everyone entering a ward can see him.

Hemorrhage within 24 hours after operation is the terror, he states, of all who look after cases of tonsillectomy. The correct treatment is to tie the blood vessel under general anesthesia. The anesthetist must be an expert. He states that bleeding from the nasopharynx is fortunately rare.

Hemorrhage after the first 24 hours after operation is seldom serious, and only once in the last ten years has he had to tie a blood vessel for this reason. The prevention of secondary hemorrhage lies in the prevention of slough in the tonsillar fossa and a help toward this is strict cleanliness in the operating theater. A final risk that must be mentioned is the mistake of leaving a swab behind. He tries to avoid this by feeling both fossae and the nasopharynx at the end of the operation and then looking into both fossae with the help of a headlight.

Five of the cases described died.

Comment: This paper is useful in that it frankly exposes the errors that were the causes of the disasters. An error recognized is an error that can be avoided. On the other hand, there are several matters of technique and of theory which are completely foreign to generally accepted practice on this continent. In our hospital more tonsillectomies are performed than in the six other large hospitals of our city together, and in the last 30 years only one such disaster has occurred.

Symonds[26] suggests that the manipulation of the head and neck under anesthesia, and especially maneuvers involving hyperextension, may be not without danger of causing serious symptoms in persons already suffering from cervical spondylosis. In support of this he cites several cases. Two of these — aged 36 and 27 years, had quadriplegia following tonsillectomy, with proof in one case of compression of the cord by a prolapsed intervertebral disc.

As the result of the cases head position during tonsillectomy has become very important medico-legally. Hence Ballantyne and MacDermott[27] examined the tracheo-bronchial tree bronchoscopically in 100 consecutive children after removal of the tonsils by dissection and the adenoids by curettage. The Boyle-Davis gag was used in each case. Three carefully described techniques were used, 50 cases in one, 25 in each of the others. Blood was found in the trachea in 12.

They state that the laryngologist is faced with two possible courses, neither of these without risk. In the first place, he can perform tonsillectomy in the hyper-extended position and run the extremely small, but equally serious, risk of paralysis from protrusion of an intervertebral disc. Such a possibility can be minimized by the exclusion, through investigation, of an existing disc lesion; but a disc lesion may be present without symptoms and accidents can also occur with a normal disc. The second course is to perform the operation in a position of less extension and to run the risk of allowing blood to be aspirated in the tracheo-bronchial tree. This hazard, though numerically much greater is obviously less serious than the first, as the blood is effectively removed by coughing when the depth — or, more correctly, the shallowness — of anesthesia is properly controlled; furthermore, most of the respiratory complications that may follow aspiration can be readily detected and efficiently treated.

They feel that a satisfactory position could probably be attained by placing the "thyroid" pillows under the shoulders and maintaining, but not in any way exaggerating, the extension so afforded by gentle support of the Boyle-Davis gag. This maneuver would aim primarily at maintaining an efficient airway; any further tilt required to prevent aspiration of blood could then be made by lowering the head of the table.

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Johnston[28] commenting on the subject believes that the cases cited by Sir Charles Symonds are a warning against hyper-extension of the neck. The reason for the extended position is to prevent blood entering the bronchial tree. Flooding of the laryngeal opening can be prevented by intratracheal *intubation with a large-bore tube.*

Godwin[29] tabulated the signs and symptoms of each of four hundred school children before and after removal of tonsils and adenoids. The average age of the children at the time of operation was eight years. The average time after operation at which the re-examination took place was one year and three months.

Before Operation		After Operation				
Signs and Symptoms	No of Cases	Cured	Percentage Cured	Improved	No Change	Percentage No Change
Enlarged cervical glands	339	200	59%	113	26	7 6%
Frequent sore throats	306	221	72 2%	77	8	2 6%
Frequent colds	110	72	65 4%	30	8	7 2%
Mouth breathing	203	—	—	13	190	93 5%
Hearing defect	48	11	23%	—	37	77%
Suppurative otitis media	13	3	23%	1	9	69 2%
Otalgia	19	11	58%	1	7	36 8%
Snoring	68	54	78 5%	7	7	10 3%
Speech defect	30	4	13 3%	19	7	23 3%
Rheumatic pains	12	2	16.6%	7	3	25%
Nocturnal enuresis	3	1	33 3%	2	—	—

Walker[30] went to the parents for opinion as to the relative success or failure of adenotonsillectomy. In this series two *very essential basic principles* were insisted upon. First, the patients were carefully selected for operation, the majority being referred for surgery after evaluation by the pediatrician, and the pediatrician was an equal partner in the prediction of good results. Second, good surgical technique as well was insisted upon.

Six sets of questions were sent out:

1. Was the operation beneficial from the standpoint of *a.* Increased resistance to infection. *b.* General well-being. *c.* Improved appetite.

A favorable response was given in 83 per cent. Some of the questions were unanswered, but even if these responses had been unfavorable an over-all percentage of favorable answers would have been 72 per cent.

2. Since surgery is any difference noted in: *a.* Number of common colds. *b.* Severity of common colds. *c.* Duration of common colds.

Answers showed that 70 per cent of operated children withstood this infection more readily. Unanswered questions, even if unfavorable, would reduce this by only 5 per cent.

3. If any of the following conditions was present before surgery please indicate the appropriate answer: *a.* Mouth breathing. *b.* Swollen neck glands. *c.* Noisy breathing at night. *d.* Restlessness at night. *e.* Constant nasal discharge in winter or year round. *f.* Cough.

Of this group of answers 89 per cent were relieved by surgery.

4. If hearing impairment was one of the indications for surgery is hearing now: *a.* Normal. *b.* Improved. *c.* Unimproved. *d.* Worse.

The answers indicated that 25 out of 28 cases were improved.

5. a. Was your child subject to ear pain before surgery? Was your child subject to ear infections with discharge?

Complete relief of otalgia was experienced in 34 out of 45 children and relief of discharge in 14 out of 22.

b. If the patient continued to have post-operative pain or discharge was there any difference noted in *1*. The frequency of pain or discharge; *2*. Severity of pain or discharge; *3*. Duration of pain or discharge.

The answers to these questions indicated that practically all in this group were less likely to have severe symptoms.

6. *a*. From a psychological standpoint do you think your child was emotionally upset by his or her hospital and surgical experience?

Out of a total of 95 answers 59 reported no effect, 24 slight effect, 7 moderate and 5 severe. Analysis of these answers indicate that the presence of parents seems to be of value in preventing psychological trauma. Of interest was one child who had both T.&A. and circumcision and suffered severe emotional trauma while his brother, who had previously undergone only the tonsil operation, was not affected. The attitude of the parents is most important. Extremely tense attitudes on the part of the parents is likely to lead to more emotional upset in the child.

b. In reference to emotional trauma do you feel that this reaction was temporary or permanent?

The answers were that reaction was permanent in three, temporary in 31 and unspecified in two out of a total of 36.

Comment: The over-all favorable results recorded in this summary are mainly attributable to proper selection of cases and good surgical technique. The selection of patients for T.&A. is much better done by the pediatrician or family doctor who is associated with the child over a period of time than by the specialist who may be consulted only once or twice at times when the upper respiratory tract is relatively full of infection.

Kenton and Parsons[31] report a new technique for anesthesia for tonsillectomy which they have used successfully on about 1,400 children, the vast majority of whom were under 10 years of age.

No mortality or morbidity occurred that could conceivably be connected with the method of anesthesia employed.

Premedication plays an important part in the success of the method, and, therefore, accuracy in its application is insisted upon. Children between four and nine years (assuming their weight to be broadly within normal limits) one hour before operation, receive 1½ gr. (0.1 g.) of quinalbarbitone ("Seconal") and 10-15 mg. of pethidine, with 1/150 gr. (0.43 mg.) of atropine by injection. Those between the age of 10 and 18 years are given 3 gr. (0.2 g.) of quinalbarbitone and 15-25 mg. of pethidine, with 1/200 gr. (0.32 mg.) of atropine. Pethidine for children is thus given at approximately 3-3.5 mg. per stone (0.47-0.55 mg. per kg.) of body weight. The well-established analgesic and antispasmodic action of pethidine when combined with the hypnotic effect of quinalbarbitone makes the induction of anesthesia by means of the sequence of inhalational agents (nitrous oxide with oxygen-trilene-ether) rapid and smooth.

With the above premedication, patients arrive in the theater fast asleep, and with judicious induction, pass from sleep, without waking up, into anesthesia within a matter of two minutes. Those who arrive in the theater conscious but drowsy are not apprehensive and readily co-operate in "blowing up the red balloon."

Induction is achieved by allowing nitrous oxide to flow at the rate of 10 litres a minutes without the mask touching the face. Sense of smell is lost in two or three breaths, and a trickle of "trilene" is added. The mask is then lowered to the face and nitrous oxide is adjusted to 6 litres, and oxygen, 2 litres, is added, the child remaining pink throughout. The expiratory valve is closed and the bag emptied two or three times to eliminate some of the lung nitrogen. Three breaths later an oral airway is introduced at the slightest suggestion of obstructed airway, and is surprisingly well tolerated; a trace of ether is then added to the mixture. The expiratory valve is now slightly opened, and the bag is very gently compressed, assisting respiration. The patient is now wheeled into the theater, and while he is being placed on the table (with-head-down tilt of about 5 degrees) and the towels are being draped in the thyroid position the ether is allowed to flow more freely; at no time need the gases bubble through the ether.

It will be found that the lower jaw is sufficiently relaxed for the Davis gag to be placed in position. The harness is not used, the gag being supported manually.

Experienced has taught us that the above technique provides adequate anesthesia for tonsillectomy and adenoidectomy in children up to the age of 10.

In children of 10 years and over greater concentration of agent or agents employed and/or intubation would be necessary to achieve the same purpose. Instead of resorting to either of the above means, we find it advantageous to cut off the access of traumatic stimuli, leaving the plane of general anesthesia light; therefore in older children the method is combined with infiltration of the vicinity of the tonsil with 1 per cent lignocaine hydrochloride without adrenaline.

A research team at Albany Medical College, Albany, New York[32] tried to ascertain whether careful preparation and handling would reduce the emotional injury sometimes experienced by children admitted to the hospital for tonsillectomy. There were 140 children, aged three to eight in the experiment, and the key people in preparing them to face operation confidently proved to be the social worker and the anesthetist. Both of these went to unusual pains and spent a great deal of time to achieve their object. Premedication was slight or not used at all. It was found that rectal temperture taking was alarming, and so, of course, were enemas. Both were given up. Injections were avoided, except for an injection of atropine just before operation, and a needle-prick for hemoglobin estimation.

The social visitor visited the child at his home ten days after the operation, and again two or three months later, to see whether he was showing any evidence of increased anxiety. She estimated that, of the 140, 30 showed favorable behavior changes. The children treated with consideration for emotional factors gave less evidence of trauma than those who were not. More than half of the 140 showed behavior that was either unchanged or improved. The younger children in the series showed more but milder adverse behavior traits than their seniors.

Over an 18-month period, Eckenhoff[33] sent questionnaires to parents of children who had otolaryngological operations, in an ateempt to find out the possible relationship between anesthesia and personality changes. The answers sought were: — *1.* Is your child a bed-wetter; since operation does he wet the bed or wet it more frequently? *2.* Does your child have night cries or terrors; since operation does he have night terrors or have them more frequently? *3.* Is your child subject to temper tantrums; since operation do they occur or occur more frequently? *4.* Has your child become afraid of meeting strangers since operation? *5.* Has your child been afraid to have his face covered since operation? *6.* Has your child become afraid of new odors since operation? *7.* How did you prepare your child for hospitalization and operation?

Replies were received from 612 or 61 per cent of the total questionnaires sent out. Seventeen per cent of the replies indicated a personality change which might be traced to anesthesia or hospital experience. The younger the child, the more likelihood there was for the development of personality changes. Premedication, when properly administered, and careful and proper induction of anesthesia lessened the chances of personality changes. It was found that bed-wetting occurs five times more commonly after the use of vinyl ether (Vin ethene) than when other techniques were used.

POLIOMYELITIS AND TONSILLECTOMY.

Lassen and Ibsen[34] gave a first-hand account of the 1952 outbreak of poliomyelitis in Copenhagen. Most of their report concerned the tremendous treatment problem which was tackled at the Blegdam Hospital, where in less than six months over 300 patients were admitted in need of special measures, such as tracheotomy and assisted respiration, by reason of bulbar or other paralysis.

In the course of this outbreak 2,722 cases of poliomyelitis were notified from among a population of approximately 1,200,000. Epidemiological investigation led to the conclusion that personal contact is an important factor in the spread of poliomyelitis.

Southcott, who has made a retrospective survey of the 1947-1948 outbreak in South Australia, finds that in 35 out of 39 cases of bulbar poliomyelitis, the patient had been tonsillectomised. This was a higher proportion than might have been expected from a random group. In only one of the bulbar cases, however, had the operation been done during the current period of prevalence; and in over half these cases tonsillectomy had preceded poliomyelitis by more than five years. Nevertheless, Southcott believes on statistical grounds that the two events were associated, and that a child whose tonsils are removed at the usual age of five to seven years, suffers trauma to the nerves of the pharynx which increases susceptibility to bulbar poliomyelitis (but not to other forms) for at least ten years.

If this finding is confirmed we shall have to regard poliomyelitis as a long-term as well as a short-term risk after tonsillectomy; and the indications for this very common operation may have to be revised. Lassen did not say whether the epidemiological investigation of the Copenhagen outbreak had been designed to examine the possible long-term association of tonsillectomy with bulbar poliomyelitis. The number of such cases surveyed by the Danish workers must be among the largest ever studied in a single locality; and this Danish outbreak seems to offer an excellent opportunity for confirming Southcott's findings in Australia.

Tolczynski[35] reviews the controversial tonsillectomy — poliomyelitis relationship by attempting to answer five important questions. For the answers he surveyed the literature and tabulated the information obtained during the severe poliomyelitis outbreak in Saskatchewan in 1952.

Question No. 1. Is the part played by tonsillectomy such as to suggest that this question increases the risk of contracting poliomyelitis? There is no unanimity of opinion on this question. Some papers would indicate that the risk is two to three times as great in the recently tonsillectomized patient. While others show that the relationship is only casual. One author was able to prove that the annual incidence of poliomyelitis in the general population is 80 times greater than in the recently tonsillectomized patients during an epidemic.

Question No. 2. Does recent tonsillectomy increase the danger of developing bulbar type of poliomyelitis? Again there was widespread disagreement with one author contending that the idea of any relationship existing belongs to the past. Others felt that the likelihood of bulbar poliomyelitis was 16 times as great in the recently tonsillectomized patient as in the general population. The preponderance of statistics would suggest, however, that in over 50 per cent of recently tonsillectomized patients who contract poliomyelitis, it will be of the bulbar type.

Question No. 3. Does the presence or absence of tonsils and adenoids influence the susceptibility to poliomyelitis, the type and the mortality of the disease? Here again there was much difference of opinion. Many authors feel that the tonsils and adenoids are a barrier to the virus and that without them patients are more susceptible to poliomyelitis and are more likely to develop the bulbar type. At the other end of the scale was a report of the 1916 epidemic in New York where there were 8,928 cases and 2,407 deaths. This report states that the disease was rare among tonsillectomized patients and affected mostly children with hypertrophied tonsils and adenoids.

Question No. 4. Should the discontinuation of the T. and A. operation be recommended during the summer months. What time then should be the proper one? Is tonsillectomy an elective operation which can be postponed indefinitely?

Most authors feel that T. and A. operations should be suspended during the summer months and especially if there is an epidemic of poliomyelitis in progress, but in certain cases where the indications are urgent the surgeon must decide whether the risk of contracting poliomyelitis is as great as the danger of delaying operation.

Question No. 5. Can a simple answer be given to the problem of polio-tonsillectomy relationship?

From his review of the literature on this question, he feels that the answer is no. There is no conclusive evidence for or against the relationship; but, because of the education of the public in respect to the danger and because tonsillectomy may precipitate poliomyelitis in children who are healthy carriers, the operation should be avoided during time of increased exposure to the virus.

Comment:

This is an interesting paper which shows the wide variation of opinions on the subject. It also shows how statistics can be found to support either extreme.

Whether or not there is some specific relationship between T. and A. and poliomyelitis it would seem wise to avoid any elective operation during an epidemic of poliomyelitis because the patient's general resistance to infection may be lowered by any operation to a degree that would render him more susceptible to an attack of poliomyelitis.

MOUTH AND THROAT INFECTIONS.

Because primary faucial diphtheria is rare during the neonatal period, Curtin[36] published a report of such a case in an infant — aged 22 days. The infant was breast fed and progressed normally until he reached the age of 20 days. At this time he became increasingly lethargic and refused to take the breast. He was pale and apathetic and had a weak cry. A gray membrane covered the left tonsil and extended backwards over the posterior pillar and also encroached upon the anterior pillar. The membrane was adherent to the underlying tissues and bled on swabbing. There was no nasal discharge. The temperature was 97.8° F. A diagnosis of diphtheria was made and 20,000 units of antitoxin were given pending the bacteriological confirmation. The report next day was C. diphtheriae and it was found to be a virulent type. The infant was admitted to hospital and a further 40,000 units of antitoxin given along with 500,000 units of penicillin. This was followed by 100,000 units of penicillin four-hourly. On the third day the infant collapsed due to vascular failure and was revived with difficulty. Two days later palatal paralysis with nasal regurgitation developed, and it was necessary to give esophageal feedings for three weeks. The infant was eventually discharged well after 164 days in the hospital.

It has long been recognized that a mother transmits diphtheria anti-toxin to her infant, but this protection may rapidly be lost. It has been suggested that the rarity of faucial diphtheria is due to the acid reaction of the mouth and the poor development of the tonsils in infancy.

The explanation in this case of faucial diphtheria was that the 14-year-old brother was a carrier of virulent gravis-type organisms. He prepared a feed of sugar and water two days before the baby fell ill, and before offering the bottle to the infant he sucked the nipple to satisfy himself that the contents were satisfactory. His own carrier state did not clear on intensive penicillin treatment but eventually disappeared following tonsillectomy.

Lall and Karelitz[37] from the Willard Parker Hospital in New York discuss the treatment of diphtheria and six diphtheria carriers with terramycin. Six cases of acute faucial diphtheria and six diphtheria carriers were studied. All cases were given 50 mgm. of terramycin per kilo per day. Those acutely ill with diphtheria were also given antitoxin shortly after admission to the hospital. The contact carriers were given the terramycin alone. Terramycin was given in all cases until three negative nose and throat cultures were obtained.

Of the six cases of acute diphthera one had negative nose and throat cultures in three days. Four had negative swabs within 24 hours and one required seven days to become negative. This is an average of 2.33 days as compared with an average of 21 days previously reported in cases who had anti-

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The results in the carrier cases were about the same as in the acute cases. The carrier state was considerably shortened. Two patients had negative nose and throat cultures within 24 hours. One had negative cultures after two days, one after three days, one after four days and one after six days.

The impression gained from this work and previous contributions by the authors was that for acute faucial diphtheria, results differ very little whether penicillin, aureomycin or terramycin is added to the anti-toxin therapy. The carrier state can be terminated by penicillin, aureomycin or terramycin in most patients. Terramycin may be given to patients who are allergic to penicillin or who harbor bacterial strains resistant to penicillin.

The condition of recurrent parotid swelling has been defined as a clinical entity by Jones.[38] Most of the published accounts refer to adults, but the disease is by no means rare in children. Seventeen cases are reported from the Midlands of England.

The essential feature of the condition is the swelling of one or both parotid glands. This recurs once or many times. In these cases recorded the whole parotid gland had been enlarged, and there was usually tenderness but no redness or edema of the overlying skin. Swelling had lasted from a few minutes to several weeks, and pain had been a variable feature. Usually there was some discomfort present. When the disease was active, muco-purulent secretion could nearly always be expressed from the affected gland. The temperature was inconsistent and was often normal. The white blood count was usually 8,000 to 10,000 but in one case went up to 24,800. In most of these cases the sialogram confirmed the diagnosis. A plain film was first taken to exclude the presence of calculus which was never found. One to 2 ml. of "Neohydinal fluid" was injected with a syringe and small blunted hypodermic needle into Stenson's Duct, and lateral and postero-anterior projections were made. In the normal gland the ducts were well outlined, and it proved impossible to avoid filling of the acini. In 10 patients of this series a spherical dilatation of the finer ducts was seen, and in others a more distinctive change occurred. In only one case was the sialogram normal.

The organisms cultured from the parotid secretion were α hemolytic streptococcus, or penumococcus, or both. The condition is thought to be due to infection in the mouth such as carious teeth and infected tonsils, or in the nasopharynx. The infection ascends the parotid duct and is associated with the stagnation of the secretion in the gland.

Treatment consisted of massage of the gland together with the local injection into Stenson's Duct of penicillin. Penicillin 100,000 units per ml. were injected daily for six days. In some cases a second course of treatment was given after an interval of several months. This treatment commonly cut the period of activity of the disease and sometimes caused complete relief.

CONGENITAL ABNORMALITIES.

In a paper on congenital choanal atresia Baker[19] found in the literature some evidence of familial tendency; also that the condition is more frequent in the female, and that 90 per cent are osseous and 10 per cent membranous. In respect to the latter figures, other investigators have found quite a fair number of mixed osseous and membraous atresias. In two-thirds or more of the cases the obstruction is unilateral.

Embryological theories as to cause are — *1.* Persistent bucconasal membrane. *2.* Persistence of buccopharyngeal membrane. *3.* Or medial outgrowth of the vertical and horizontal processes of the palate bone. ,

Clinically this condition should be suspected in all cases of asphyxia neonatorum, especially if the infant has repeated attacks of cyanosis or suffocation which are definitely relieved by coughing and aggravated by nursing or sleeping.

Diagnosis can be made by failure of a probe to pass into the nasopharynx, or in older patients additional help may be obtained by X-ray, using lipiodol. There is always a considerable amount of thick glistening mucus on the floor of the nose.

Surgical correction has been done intranasally, trans-septally, transantrally, and also by the transpalatine approach.

He feels that, while the intranasal approach is successful, it is not too satisfactory, because it requires a great deal of time and aftercare on the part of the surgeon and brings considerable discomfort to the patient. The transpalatine procedure will possibly be the operation of choice in the future.

Ersner[40] reported a case of bilateral bony choanal obstruction in an eight-year-old girl, which he treated by removal of the bony obstruction by operation through the nose. The openings were kept patent by inserting a No. 26 French catheter in the form of a rubber doughnut. This was changed to a No. 22 after three weeks, and the latter was left in until 48 days post-operatively. Thereafter there was no tendency to close, and the nasal respiration was perfect. It was interesting that, with establishment of air currents through the nose, the child was able to smell things and taste her food for the first time.

Bilateral choanal atresia must be included in the differential diagnoses of all cases of asphyxia neonatorum.

He suggests that, when adenoids are removed, some form of nasal sound should be passed to ascertain patency. He has discovered five previously unsuspected unilateral choanal obstructions with this routine.

The Treacher Collins' Syndrome is constituted by the following congenital abnormalities.

1. Bilateral notchings of the lower eyelids.

2. Deficiency of the malar bone and infra-orbital ridge.

3. Lack of cilia on the medial third of the lower lids, with oblique palpebral fissures.

4. Micrognathia.

5. Bilateral deformities of the ears.

6. Absence of the puncta of the lower eyelids.

They mention also several less frequently occurring congenital defects, the most important of which are:

7. Nasal deformities.

8. Malocclusion.

9. Absence of the external auditory meatus with degrees of deafness.

10. Cleft lip and palate.

11. Deformities of the extremities.

McNeill and Wynter-Wedderburn[41] described a three-months-old child who had a number of the above deformities and in addition bilateral choanal atresia. Illustrated by four photographs.

Finerman and Pick[42] discuss the problem of nasal encephalomeningocele. There are three main groups. Occipital—these are the most common. Sincipital—externally visible tumors of the nose or forehead or the medial corner of the eye or anterior part of the orbital cavity. Basal herniations are not externally visible and can be subdivided according to whether they come—

1. Through the sphenoid bone or between the sphenoid and the ethmoid when they are referred to as Encephalomeningocele Spheno-Pharyngea.

2. Through the cribriform plate to produce an intranasal tumor.

3. Through the supraorbital fissure into the orbit behind the eyeball.

4. Through the supraorbital fissure and on down via the infraorbital fissure into the sphenomaxillary fossa producing a tumor on the medial side of the mandibular ramus.

In one report of 49 cases of intranasal encephalomeningocele only 20 cases survived treatment. Most succumbed following surgery from immediate meningitis or later meningitis due to an upper respiratory infection.

Inclusion of encephalomeningocele in the differential diagnosis of intranasal tumors is, therefore, more than academic. The following are some diagnostic criteria: —

1. A nasal tumor found at birth or symptoms of nasal obstruction found in early childhood.

2. A widened nasal root or unusually large pupillary distance.

3. The presence simultaneously of an external encephalomeningocele.

4. A tumor arising high in the nose over which the septal mucous membrane appears to continue.

5. Evidence of intracranial communication such as fluctuation synchronous with the pulse or respiration.

6. Post-operative cerebro-spinal rhinorrhea.

X-ray is of little aid even with defects up to one centimeter in diameter. Diagnostic puncture or biopsy are dangerous. The best diagnostic aid is to suspect the possibility of such as anomaly.

Dolowitz, Rambo and Stephens[43] report a study of family in which hereditary hemorrhagic telangiectasia has occurred in the past six generations. Eighty-three cases occurred out of 491 members reported on. The disease behaves as a Mendelian dominant. Pathologic changes are confined to the blood vessels and the effects of hemorrhage. The basic lesion is a greatly dilated endothelial space which is deficient in elastic and muscular components. Although attention is usually centered on local manifestations careful search may show the lesions to be widespread.

Attempts to prevent hemorrhages have been most unsatisfactory, because, although it is easy to destroy the primary lesion by cautery, X-ray or some other method, satellite lesions soon form several millimeters away; therefore, control of hemorrhage is about all one can do, and in this regard the excessive fragility of the telangiectatic vessels must be recognized. Oxycel or gelfoam or similar topical hemostatic agents are the best methods because these materials do not require removal. The clot, therefore, is not disturbed and secondary hemorrhage is frequently averted.

SURGERY.

Jordan[44] has written on the importance of early diagnosis and treatment of nasal fractures.

Rather than wait until swelling has subsided by natural means, the edema can be dispelled immediately by the injection of hyaluronidase into the tissues. This is a specific enzyme which hydrolyzes the intercellular cement known as hyaluronic acid with the resulting hastened diffusion of tissue fluids.

He uses mainly local anesthesia in adults and uses the usual intranasal cocaine and adrenalin plus injections of a solution made up in the following proportions: 150 units hyaluronidase and 0.5 cc. of 1:1,000 adrenalin to 25 cc. of one per cent procaine. He recommends similar measures in children with the addition of general inhalation anesthesia.

He describes his methods of treating nasal fractures and points out that open reduction is often advisable by the rhinoplastic methods of Foman. Such open reduction has the advantage of: *1.* Accurate diagnosis by inspection. *2.* Evacuation of hematoma and better drainage. *3.* Precise reduction under direct vision. *4.* Correction of pre-existing defects. *5.* Attention to associated fractures. *6.* The lessening danger of post-operative deformities and impairment of function.

Following reduction adequate intranasal packing and a rigid external dressing help to maintain position. With continuous protection of antibiotics the packing can be left in as long as seven days.

Cottle, Quilty and Buckingham[45] present some of their experiences with different types of material in the attempt to improve the appearance and restore the function of variously deformed noses.

The most specific needs for implants in children are: *1.* The restoration of contour of the roof, especially over the cartilage vault; *2.* The stretching of the skin in this area in preparation for future permanent correction; *3.* The prevention of subcutaneous cicatrix formation in the dorsum region and, *4.* The repair of septum deformities and defects.

For all of these they have used preserved human and ox cartilage with about equal results. Ten to 20 per cent absorption of the grafts is to be expected . . . Small implants of plastics and ivory are remarkably well tolerated. To obtain good-sized straight non-curling slices of cartilage, we used the crest of the ilium in children. In the very young the iliac crest is cartilaginous — not cancellous and cortical bone, and slices are easily obtained without endangering the future growth of the pelvis. The fate of this cartilage is no surer or different from other autogenous or preserved cartilage. It is as readily absorbable, but they have never seen it start an appreciable local reaction.

The care of hematoma and abscesses of the septum especially in children requires special attention.

Following the usual drainage instituted in such cases a careful inspection of the septum must be made to determine the extent of the septal cartilage destruction. It will often be learned that much if not all of this quadrilateral cartilage has disappeared and perhaps, too, the upper lateral cartilages are involved. This leads to saddling of the cartilaginous vault, retraction of the columella and deformities of the upper lateral cartilages, and the nasal lobule. Replacement of the missing cartilage must be made.

Autogenous cartilage from the ribs, ears, or crest of the ilium can be used. In their experience all of these as well as all other substances are very likely to be absorbed or extruded.

They feel that following an abscess or hematoma the septal tissues are not ready to receive implants for many months, but at the same time if they allow healing to proceed unhindered the fusion of the two mucosal flaps will become so firm with solid scar formation that subsequent separation will be impossible; therefore, they recommend that at about 8 to 12 weeks following subsidence of the acute infection some material be placed between the flaps even though the benefit may not be permanent. For this they have used autogenous or preserved cartilage. Not one was permanently retained; however, one to two years later they have been able, in all of their patients, to find a plane of separation and have then reinserted the implants. The value of ox cartilage in these instances has been most noteworthy. One of their ivory implants in an adolescent septum is still intact after 13 years.

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FIFTY-SECOND CONGRESS FRANCAIS d'OTO-RHINO-LARYNGOLOGIE.

The 52nd Congrèss Francais d'Oto-rhino-laryngologie will meet October 18 to 22 in the Grand Amphithéâtre of the Faculté of Médecine, Paris, under the presidency of Professeur J. Terracol, of Paris, and honorary president Professeur Lèon Binet, member of the Institut, Doyen of the Faculté of Médecine of Paris. A number of interesting papers will be given, among them, "La Thérapeutique par les ultra sons en Oto-rhino-laryngologie," by Professeur Portmann, with MM. Michel Portmann and Louis Barbe, as collaborators; "De la greffe cutanée libre en chirurgie otologique," by MM. Ombrédanne, Clerc, and Poncet.

LATERAL PHARYNGEAL SPACE DISSECTION AS AN ADJUNCT IN RECONSTRUCTING THE MUSCULAR VELOPHARYNX.*

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One of the most controversial surgical problems of all time is the reconstructive management of clefts of the muscular velopharynx. As the investigator plods through the voluminous literature on this subject and listens to innumerable contradictory dissertations regarding it, he is likely to reach a state of total confusion. Perhaps the simplest method of reorienting himself is to review the historical panorama of surgical exercises and to pick out in chronological order those which *have the most practical application. To assist in this* endeavor we feel that he should lean heavily on the splendid research done during the past two decades.

HISTORY.

Though Albuscasis of Arabia wrote on deformities of the mouth as early as the eleventh century,[1] medical literature contains no clear reference to the congenitally cleft velum prior to 1739, when Sharp casually mentioned that it was relatively common to find "the roof of the mouth fissured."[2] A French dentist, Le Monnier, was first to report a successful repair,[3] and it was not until the nineteenth century that surgical cognizance was taken of this malformation.[4] In 1816 von Graef described an effective surgical operation,[5] but it remained for Roux to popularize staphylorrhaphy as the result of a much-publicized operation on a Canadian student.[6] This event marked the beginning of a long period of surgical experimentation.

The cleft velum presents the surgeon with an unusually complex problem. Closure must be accomplished and physi-

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ological function must be established in such a manner that normal growth patterns are not thrown out of balance, and the face may continue to develop in proper proportion. Closure itself absorbed all the attention of the early workers. No effective knowledge of regional anatomy, or of the physiology of speech, existed to govern approach to a solution; therefore, it is not surprising that while some of the procedures advocated were merely variants of one another, others were diametrically opposed.

Almost from the beginning there was recognition of a need to counteract the lateral muscle pull that interfered with healing after staphylorrhaphy. Different kinds of metal sutures were used and several types of relaxing incisions were attempted. Froriep, in 1823, was first to sever the palatal muscles, and ten years later Dieffenbach also advocated cutting these muscles to obtain relaxation.[7] Incisions for that purpose then came into frequent use. Such incisions divided various muscles, as dictated wholly by chance, until in 1844, when Fergusson was able to present a careful dissection of the muscles of the cleft velum and a classic description of their function.[8] Fergusson himself practiced myotomy, with successful closure in 129 of the 134 cases he operated on between the years 1828 and 1864.[9] His method, or a modification thereof, was popular for a generation. Seeking a more satisfactory method of relieving tension, Billroth in 1861 suggested fracture of the hamular processes, and later vigorously deplored myotomy because of its resultant scarring and immobilization.[10]

In the meantime, another important forward step had been taken. Von Langenbeck observed bone regeneration in a resected maxilla with intact periosteum, and was thereby motivated to use the palatal periosteum in flaps mesially displaced from each side of the split palate.[11] In this manner the blood supply to the flaps was theoretically assured, and a second basic cause of failure in closure was thought to be overcome. Later von Langenbeck greatly refined his procedure, utilizing the best features of each of the several methods previously employed.* The success of his efforts earned for him the honor of being both approved and imitated by his contemporaries.

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* Dorrance calls him "the codifier of cleft palate surgery" (loc. cit., 9).

During the latter part of the nineteeth century, knowledge and practice had so far advanced that successful closure was generally achieved by the skilled surgeon; however, Passavant in 1862 called attention to the nasal twang resulting from palate repair, and attempted several means of improving functional results.[12] Fergusson and others then turned their efforts toward that goal, but there was still no clear understanding of the intricacies of the problem.

The first detailed account of the speech defects associated with clefts of the palatopharyngeal sphincter was written by J. A. Bond in 1893.[13] He emphasized that patients suffering from such a congenital malformation should have speech training subsequent to operation. Sir James Berry was probably the first surgeon to publish a detailed analysis of the speech results in a series of repairs.[14] Thereafter, attention was concentrated on speech improvement, and great strides were taken by the early twentieth century surgeons toward obtaining better functional results. Ganzer,[15] Veau,[16] Dorrance,[17] Turner,[18] and Wardill[19] were the leaders in this surgical endeavor. Morley's excellent treatise carried this work much further,[20] and there is promise of additional development when the photographic research done by Kemper and colleagues is published.*

Slowest to yield to advancing surgical knowledge was the problem of maintaining proportional development of the face after palate repair. Bone growth studies had been made by Sir John Hunter with a series of madder feeding experiments late in the eighteenth century but were not yet correlated with the surgical problem of reconstructing the split palate.[21] Ernst was perhaps first to appreciate their importance in that respect. Anticipating later studies, in 1926 he wrote:

> *"Each early operation has the effect of retarding the development of the jaw of the child, because the vital forces do not stand in proportion to those created by sur-*

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* Personal conversation with John W. Kemper, Sept., 1949.

> *gical intervention, such as the suture, the resulting scar, and scar tissue."*

His investigations led him to conclude that in patients with clefts of the velopharynx, the mesopharynx is wider than in those without the malformation and that the halves of the maxillae are not normal in size, but smaller, He added:

> *"If one strives to eliminate obturators, then a circular narrowing of the pharynx is required to such an extent that the function of the muscles is sufficient to close the opening between the nose and mouth. In most cases, such a narrowing of the pharynx is sufficient."*

Ernst also wrote of the retroposing technique which Ganzer had described in 1917.[11]

Ernst's recognition that the mesopharynx in this malformation is widened in both the anteroposterior and the lateral diameters and that the musculature of the palatopharynx must be re-assembled into a position where it can function physiologically, should have played a most important part in all subsequent plans of surgical attack.

It is the purpose of this paper to review the anatomy of the region, to point out a natural anatomical route to the cleft velum, and to describe a procedure whereby the maximum physiological function can be obtained with minimum trauma and minimum interference with the growth processes.*

In our hands, use of this method has been responsible for more normal growth patterns of the face, and for a considerable improvement in the physiological function of phonation, in a series of 120 cases operated on since 1945. Ages of patients in this series have ranged from three and one-half years to 50 years.

ANATOMY.

The palatopharyngeal muscular mechanism is an extremely intricate one, so intricate that students of speech production are still baffled by some of its more complicated patterns. One readily recognizes its complexity upon stroboscopic examination of this highly-integrated muscle group; therefore, a profound responsibility is placed upon the surgeon who attempts the task of rebuilding its essential elements into such a physical state that they will perform as normally as possible. This objective is not attained by the mere assembling of the inert tissues of the palate to form an inactive diaphragm between the mouth and the nose; it is accomplished only by a direct surgical approach to the essential muscular structures that comprise the palatopharyngeal sphincter in its entirety.

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* A 26-minute sound and color film, including both pre- and post-operative speech recordings and complete operative procedure, is available upon request.

Fortunately for the surgeon, the muscle groups which make up the normal velum and those of the split velum are essentially the same. This is also true of the nerve and blood supply. However, the split velum presents two congenital anomalies which make the difference between good speech production and the distorted patterns associated with clefts of the velum: first, a widened bony and muscular pharyngeal vault containing two separate anatomical entities which cannot act physiologically until built into one single mechanism, a palatopharyngeal sphincter; second, degenerative changes, including atrophy and hypoplasia, of all or part of the soft tissues and skeletal structures comprising the palatovelar architecture. These changes begin when the halves of the palate fail to unite in intra-uterine life and continue because of distorted physiology. Only by surgical rehabilitation can these processes be halted.

The musculature of the palatopharyngeal sphincter, together with its nerve supply, lies within the dome of the pharyngeal tube. With the exception of the tensors, the sphincter is almost completely enveloped by two of its most important components: the superior constrictors of the pharynx. In enumerating its anatomical boundaries one has only to outline those of the superior constrictors (see Fig. 1). Above, the constrictors are literally hung from the base of the skull (see Fig. 2). They are bounded laterally by the pharyngomaxillary fossae, or lateral pharyngeal spaces (see Fig. 3). Their points of origin include the posterior border of the hor-

Horizontal section through head at level of angle of lips demonstrating anatomical relation of superior constrictor of pharynx and surrounding lateral pharyngeal space. (right)

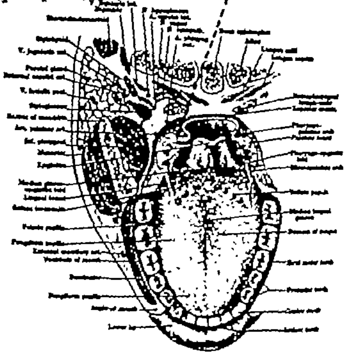
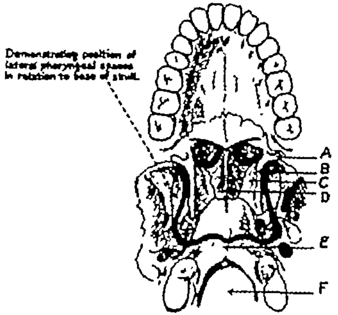


FIG. 1.


Demonstrating position of lateral pharyngeal spaces in relation to base of skull.
A
B
C
D
E
F

The heavy line shows the attachment of the fascia pharyngobasilaris to the base of the skull. A, Indicates the hamular process; B, pterygoid plate; C sphenoid bone; D, vomer; E, occipital bone; F, foramen magnum.

FIG. 2.

izontal plate of the palatine bones, the lower third of the medial pterygoid plates and their hamulae, the pterygomandibular ligaments, the alveolar processes of the mandible, and fibers from each side of the tongue.

Because of the anatomical architecture of the pharynx, it is readily seen that by freeing the superior constrictors from their bony ligamentous, and musculo-fascial attachments and by dissecting free their surrounding lateral pharyngeal spaces, complete mobilization of the muscular components of the velopharyngeal sphincter may be attained.

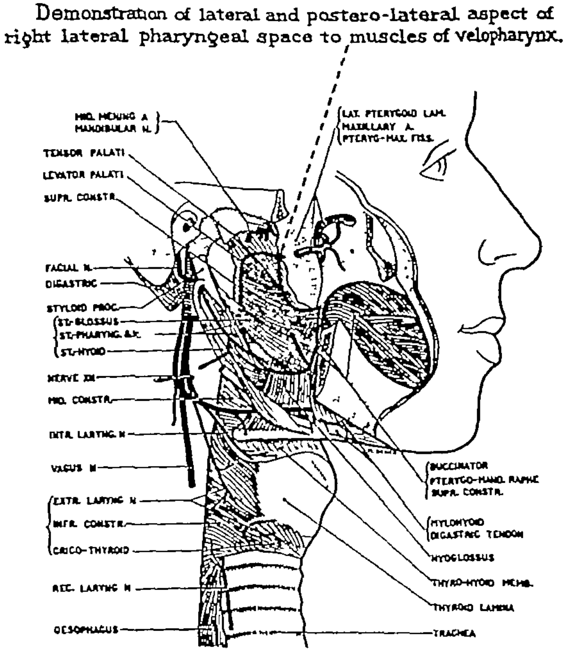
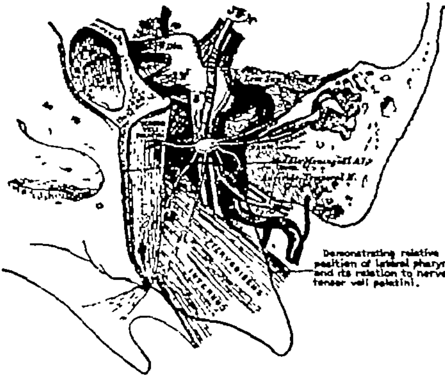


Fig 3

The entry to either lateral pharyngeal space is made by splitting one of the most important ligamentous suspensory attachments of the velum, namely, the pterygomandibular raphe. Once this is done, the antero-superior aspect of the lateral pharyngeal space lies open so that the rest of the space can be ablated by blunt dissection. By this method the nerve supply to the tensor, which enters that muscle in its upper third, is not cut or even traumatized (see Fig. 4). The same may be said of the motor innervation of the remaining muscles of the velum. Fig. 5 demonstrates the exit of the glossopharyngeal, vagus, and accessory nerves from the carotid



Demonstrating relative anatomical position of lateral pharyngeal space and its relation to nerve supply of tensor veli palatini.

The otic ganglion and its branches.

Fig. 4.

Demonstrating posterior dimension of lateral pharyngeal space, showing its relation to motor nerve supply of muscles of velopharynx.

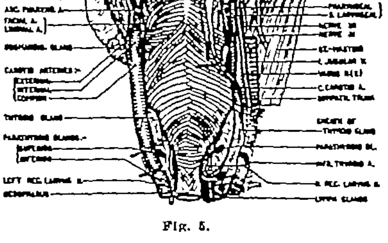


Fig. 5.

sheath, high in the neck and posterior to the lateral pharyngeal spaces. Careful dissection eliminates any possibility of interfering with the function of these nerve elements which make up the pharyngeal plexus.

In dissecting the lateral pharyngeal spaces one should keep in mind that the blood supply in this area is composed of two vessels: One is the ascending palatine artery, which traverses vertically upward over the superior constrictor and occupies an anterior position in the fossa. The other is the ascending pharyngeal, which lies over the muscle and is also vertically directed upward, but in a posterior position. In our experience, careful manipulation of the tissues prevents damage to these vessels and thereby obviates excessive bleeding.

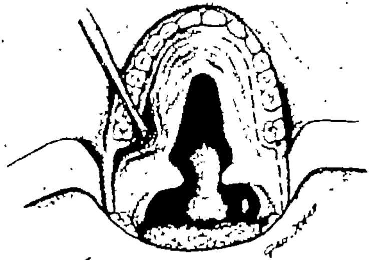
AGE OF PATIENT AND TYPE OF ANESTHETIC.

Candidates are preferably between the ages of four and one-half and six years. In our opinion, all of the deciduous teeth should have erupted and the bite be well established before surgery takes place. In some instances we delay until the six-year molars have begun to erupt. By following this pattern of attack we are more nearly certain that any surgical interference about the pterygoid abutments will not tend to retard or stop further growth processes about the mid-third of the face.

Having determined that the patient is physically fit for surgery, either hypopharyngeal ether insufflation or an endotracheal anesthetic is administered. In addition we have routinely used a local anesthetic, composed of one per cent novocaine with 20 drops of adrenalin to the ounce, to infiltrate the soft tissues of the hard palate and the lateral pharyngeal spaces. The use of this local anesthetic accomplishes a dual purpose: it allows the anesthetist to carry the patient in a lighter plane of anesthesia, and by providing a dried field it assists the surgeon to perform lateral pharyngeal space dissection with greater ease.

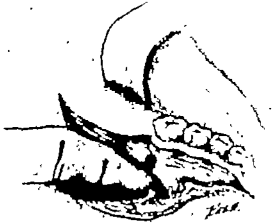
SURGICAL PROCEDURE.

Fig. 6 demonstrates the lines of incision chosen for the surgical dissection. The tissues of the hard palate are elevated in their anterior extremity and the dissection is continued in



Lines of incisions made.
Beginning of dissection.

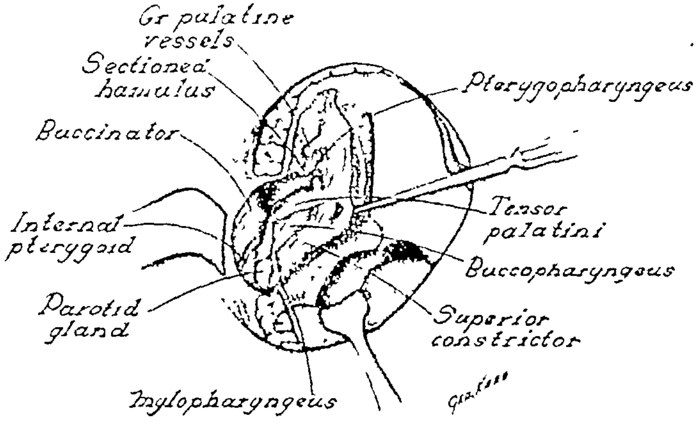
Fig. 6.



Showing blunt dissection of pharyngo maxillary fossa after hamulus has been sectioned

Fig. 7.

a posterior direction. After identifying the greater palatine vessels, the dissection is extended posteriorly until the pterygoid hamulus is exposed and severed. If at this point the pterygomandibular ligament is split from before backwards, the dome of the pharyngomaxillary fossa, or lateral pharyngeal space, will be opened, and easy access may be gained to perform blunt dissection of the fossa (see Fig. 7). By doing so, the component "muscle block" of the velopharynx on the dissected side will be freed, so that when the rest of the dissection is completed it will assume a relatively normal anatomical position. Following dissection of the fossa, the attachments of the superior contrictor to the posterior border of the horizontal plate of the palatine bone and the lower third of the medial pterygoid plate are severed. This maneuver allows the surgeon to elevate the greater palatine vessels from their bed in the pterygopalatine canal, so that when transfixation takes place an adequate blood supply is assured (see Fig. 8).



Direct approach to mesopharynx by opening the pharyngomaxillary fossa

Fig. 8.

With the lateral dimension of the muscular velopharynx dissected free, it has been our plan not only to cross-hatch but also to de-epithelize completely the mesial surface of this muscle group (see Fig. 9); then upon transfixation the split blocks of muscles can unite into one integral unit which, after healing, can function physiologically.

Having completed dissection of one side of the velum, the lateral pharyngeal space is gently packed with dry gauze and the contralateral side is handled surgically in the same manner.

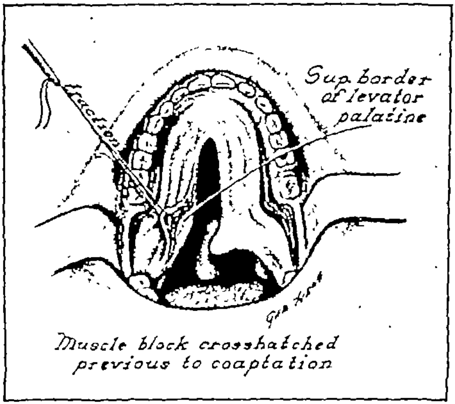

Traction
Sup. border of levator palatine
Muscle block crosshatched previous to coaptation

Fig. 9.

As the dissection of both sides becomes complete and the anatomy of the velopharynx is brought into view, anatomically there are presented two independent muscle groups. Each is made up of: *1.* The tensor, laterally; *2.* The superior constrictor of the pharynx; *3.* The levator; *4.* The salpingopharyngeus; *5.* The palatopharyngeus; *6.* The palatoglossus.

We have termed these groups *"muscle blocks,"* for in reality they constitute two highly-integrated blocks of muscles which heretofore have acted independently. By transfixing and uniting them in their normal anatomical habitat, good velopharyngeal function will result.

When the dissection of both halves of the velum is completed and the lateral pharyngeal spaces are opened widely, it becomes apparent that the whole muscular velum can be transfixed through the lateral pharyngeal spaces (see Fig. 10). Working in these anatomical spaces permits the surgeon to unite more perfectly the two muscle blocks that are eventually going to form a functioning palatopharyngeal sphincter. For this purpose we have routinely used through-and-through sutures of No. 0 chromic catgut. Fig. 11 demonstrates the coapted and sutured mucous membrane of the ventral and dorsal surfaces of the velum. With the muscular velum transfixed, the lateral pharyngeal spaces stand wide open. It has been our practice to simply pack them lightly with Oxycellulose gauze to control any oozing which may occur following surgery. These spaces are not touched afterward and usually have healed by granulation within a period of two weeks following the operation.

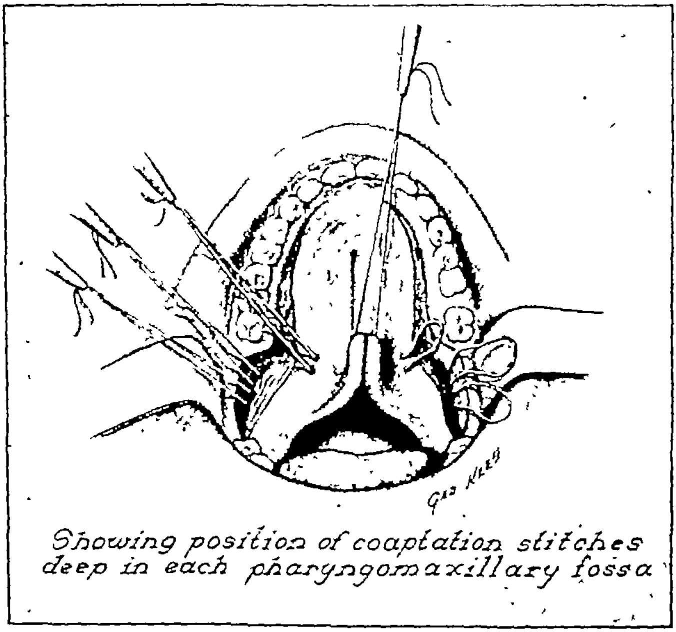
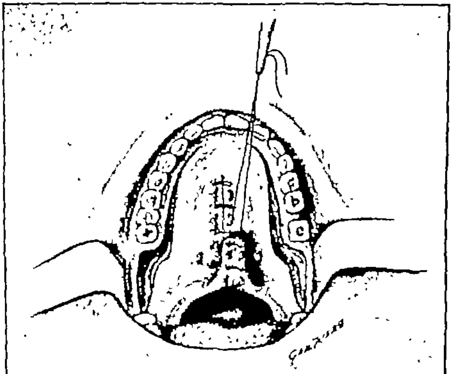
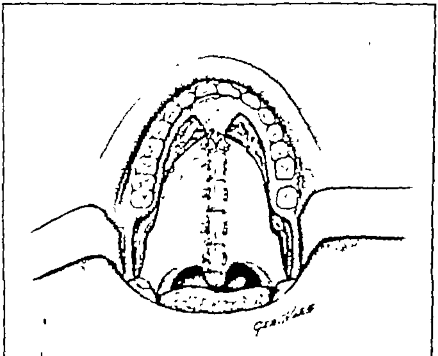

Showing position of coaptation stitches
deep in each pharyngomaxillary fossa

FIG. 10.



Demonstrating suture line of ventral surface of velum

Fig. 11



Setback manuver used when palate is congenitally short

Fig. 12.

We have consistently used lateral pharyngeal space dissection when doing retroposing procedures of the velum. In dealing with congenitally short palates, or those presenting a sub-mucous cleft, we have found that this method lends itself admirably to the transposing technique. The same principle also applies to those cases which have been previously operated on with failure and which contain considerable scar tissue, with shortening of the velum (see Fig. 12).

SUMMARY AND CONCLUSIONS.

A review of the literature has been done to determine those methods which have most practical application to the reconstruction of the palatopharyngeal sphincter. An attempt has been made to throw into sharper focus the importance of the anatomical position of the muscles, nerves, and blood supply of the velopharynx and its surrounding pharyngeal spaces. It has been our experience that by a more complete anatomical understanding of these structures, the surgical approach to their reconstruction through the lateral pharyngeal spaces has assured us of functional results that far surpass those obtained in the past. These conclusions are based on results in a series of 120 cases of lateral pharyngeal space dissection.

APPENDIX.

CHRONOLOGY OF DEVELOPMENT IN PRACTICAL TREATMENT OF SPLIT PALATE AND VELOPHARYNX.

1013-1106 Albuscasis, Arabian physician, was first to write on deformities of the mouth (Garrison, 131-132).

1739 Sharp wrote of cleft palate as "not uncommon" (Dorrance, 3).

1764 Le Monnier, French dentist, reported first successful repair of cleft velum (Padgett, *Surgical Diseases*, 391).

circa
1770 Sir John Hunter began studies of bone growth (Wylie).

1779 Eustache *proposed* suture of velum to Acad. Med., Paris, and was first to call attention to difficulties in deglutition and speech in cases of both surgically split velum and congenitally absent velum (Dorrance, 3).

1817 Von Graefe did first successful surgical repair of congenitally split velum (Morley, 28; Padgett, *Surgical Diseases*, 391).

1819 Roux of Paris successfully operated on Canadian student (Dorrance, 4).

1820 Warren of America did first successful repair in this country (Padgett, *Surgical Diseases*, 391).

1823 Froriep was first to sever palatal muscles *(Ibid).*

1826 Dieffenbach did osteotomy on horizontal plates of maxillary process (Garrison, 499-500).

1827 First to attempt to close cleft by flap dissected from palate itself (Morley, 31).

1828 John Collins Warren, Boston, introduced operation of staphylorrhaphy for fissure of soft palate (Garrison, 499-500).

1828 Dieffenbach first suggested separation of soft parts of palate from underlying bone (Morley, 28).

1829 Dieffenbach closed defect in palate by use of tissue from cheek (Dorrance, 200).

1834 *Dieffenbach did first successful closure of both hard and soft palate*; used lateral incisions; advocated myotomy (Padgett, *Surgical Diseases*, 391).

1837 Mettaur, in America, relieved tension by several small incisions in various parts of palate (Morley, 28).

1842 John Mason Warren closed hard and soft palate successfully; is sometimes credited with being first to do this (Garrison, 503). He *did not use lateral incisions (Padgett, Surgical Diseases, 391).*

1844 Fergusson gave first classic description of function of palatal muscles in cleft palate (Dorrance, 7).

1845 Fergusson advocated severance of palatal muscles (Padgett, *Surgical Diseases*, 391).

1853 Baizeau claimed originality for Dieffenbach operation *(Ibid).*

1861 Von Langenbeck included periosteum in flaps (Smith, 44).

1861 Billroth suggested fracture of hamular processes to relieve tension; also claimed originality for Dieffenbach operation (Padgett, *Surgical Diseases*, 391).

1862 Von Langenbeck described median suture in detail (Morley, 29).

1862 Passavant called attention to nasal twang in speech after closure of palate and proposed corrective procedure (Smith, 44); Morley, 34).

1868 Passavant described contraction of superior constrictor muscle on posterior pharyngeal wall (Morley, 36).

1870 Tait extended lateral incisions over alveolar ridge whenever necessary to gain greater width of flaps; recognized importance of tensor palati muscles (Smith, 44).

1873 Fergusson advocated osteotomy for relaxation (Padgett, *Surgical Diseases, 391).*

1877 Lannelongue, first to use mucosal flap from septum for repair *(Ibid).*

1889 Billroth deplored lateral incisions (Smith, 45; Morley, 37); he suggested breaking off mesial ptergoid plate (Dorrance, 383).

1890 Davis-Colley described "criss-cross" operation for closure of hard palate (Dorrance, 121-124).

1893 Bond gave first detailed account of speech defects associated with clefts of the palatopharyngeal sphincter (Morley, 38).

1897 Sir Arbuthnot Lane described closure by flap dissected from one side of palate (Morley, 31).

1901 Von Eiselberg repaired cleft with flap from forearm (Morley, 31).

1905 Sir James Berry published detailed account of speech results in his series of operations (Morley, 40-41).

1911 Blair closed palate with flap from neck (Morley, 31).

1917 Rosenthal used flap from upper arm for closure of palate *(Ibid).*

1920	Ganzer described method to produce longer velum (Morley, 41; Dorrance, 206).
1921	Gillis and Fry performed staphylorrhaphy alone and closed the opening in the hard palate with obturator, thereby destroying the insertion of some palatal muscles (Smith, 45; Morley, 42).
1922	Veau analyzed anatomical and functional results of staphylorrhaphy. He advocated a procedure differing entirely from that of von Langenbeck. He "elevated and displaced medially flaps of mucoperiosteum with a single posterior base . . . ". Smith calls his work "monumental" (Smith, 45; Morley, 42).
1923	Brophy described compression method of closure (Morley, 33).
1925	Dorrance presented the "push-back" operation to lengthen and relax the velum (Smith, 45-46; Dorrance, 403-439).
1926	Ernst published his conclusions that the mesopharynx is wider and the halves of the maxillae smaller than normal, in patients having cleft velopharynx (Ernst).
1927	George Grey Turner reported functional results in a series of cases. Using Langenbeck-Fergusson operation he found little improvement in speech in children under one or two years of age (Morley, 44-46).
1928	Wardill reviewed results of cleft palate surgery and concluded that closure was usually achieved by skilled surgeons, but restoration of normal speech was rare, with little improvement in procedure during the past century. He advocated a two-stage operation, based on recognition that in cleft velum the bony nasopharynx is increased in size (Smith, 46-49).
1932	Denis Browne described a two-stage modification of the Langenbeck operation (Morley, 52-54).
1934	Invention of Broadbent-Bolton roentgenographic cephalometer (Wylie).
1937	Wardill presented improved procedure having the following advantages: (1) It is completed in one session, (2) Nasopharyngeal valve action is practically assured, (3) It is applicable to all types of cleft, (4) The V-Y flaps assure maximum length with minimum scar formation, (5) If performed before the speech age, most children will learn to speak naturally without special training," according to Smith (p. 49; see also Morley, 56-59).
1946	Padgett described pharyngeal flap operation (Conway).
	Kemper and colleagues did photographic research on patients in whom a clear view of the superior velar surface and the entire nasopharynx is visible; results may revise our present opinion of muscular action during the production of speech (Smith, 49).

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BRONCHOESOPHAGOLOGY COURSE.

The next Bronchoesophagology Course to be given by the University of Illinois College of Medicine is scheduled for November 8-20, 1954. Under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

ANATOMY AND HISTOLOGY OF THE EXTERNAL EAR IN RELATION TO THE HISTOGENESIS OF EXTERNAL OTITIS.

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and
BEN H. SENTURIA, M.D.,*†
St. Louis, Mo.

The present report is a summary of certain basic anatomic information regarding the external ear, particularly concerning its blood supply and innervation. A series of histological observations has been made of normal ears in order to extend this anatomic information to include microscopic detail. The eventual purpose of the anatomic and histologic synthesis is to shed light upon the histogenesis of inflammatory phenomena and their sequelae, which are seen in the external ear under a variety of clinical circumstances[1] and to detect, if possible, any conditioning influence exerted in this region by the peculiar nature of its structure.

The external auditory meatus is lined with stratified epithelium, which usually shows some degree of keratinization. The corium or dermis is variable in thickness in different parts, being thicker where the larger and more numerous apocrine glands are grouped. The epidermis invaginates the dermis to form the sheaths of the hair follicles, which are short, irregularly dispersed, and less numerous in the inner third of the canal than in the outer two-thirds.

The glands found in the dermis are of two kinds: superficially and mainly attached to hair follicles are the sebace-

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ous glands; deeply placed in close relationship to the cartilage and the vessels near it are the tubular coiled glands, sometimes called ceruminous but more exactly referred to as apocrine glands.[2]

The ducts of the apocrine glands are long and communicate with the pilo-sebaceous units for the most part, although some of the ducts are branched and empty directly through the epidermis.[3,4] The size of the apocrine glands is considerable, ranging from 0.5 to 2.0 mm. They are most numerous in the middle third of the canal but are also found on the superior wall close to the tympanum in small numbers.

The ducts of apocrine glands are twisted and curved, are *relatively long and have a variable diameter, averaging about* 30μ.[5] The glandular epithelium is tall, cuboidal, with a domed free border. Its cytoplasm contains lipoid and pigment granules which are sudanophilic and fluorescent.[6] There is a good deal of variation in size and secretory content of the cells, depending upon their activity.[7] Elongated contractile cells, somewhat larger in the ducts, form a basement layer, which is uniformly present.

THE VESSELS AND NERVES OF THE AURICLE AND EXTERNAL AUDITORY CANAL.

Arteries and Veins. The arteries of the auricle and of the external auditory canal arise from the temporal and internal maxillary. The anterior surface of the auricle and the external portion of the meatus are supplied by the anterior auricular branches of the superficial temporal artery. A branch of the posterior auricular artery, arising from the external carotid, supplies the posterior surface of the auricle. The branches of these two arteries have extensive anastomoses. The blood supply to the deeper portions of the external auditory canal is provided by the deep auricular artery, a branch of the internal maxillary. The anterior, posterior and deep auricular veins generally empty into the external jugular and mastoid veins; a portion of the veins of the external ear empty into the temporal and internal maxillary veins.

Some of the smaller branches of the arteries and veins pierce the membrane which roofs over the cartilaginous meatus. Others traverse clefts in the anterior cartilaginous wall, the fissures of Santorini, and others the fibrous connective tissue which unites the cartilaginous with the osseous meatus. They then ramify in the lining membrane of the auditory canal and form a fine capillary network on the perichondrium. A vascular bundle of considerable size extends along the superior wall of the meatus to the superior periphery of the membrana tympani and passes on to the membrane.

Lymphatics. Politzer[8] and Rouviere[9] have pointed out that the lymphatics of the external auditory canal are probably in close connection with the lymphatics on the mastoid process and the parotid gland. Politzer observed that in inflammatory affections of the meatus, the lymphatics in the neighborhood of the ear become enlarged. The lymphatics of the anterior and superior walls of the meatus, the tragus, and the surrounding parts empty into the preauricular glands (on the parotid), those of the lobule, the helix, and the inferior wall of the meatus, empty into the infra-auricular glands (in the angle of the jaw); those of the anti-helix and the concha empty into the mastoid glands (on the apex of the mastoid process), and those of the posterior wall of the meatus, together with those of the Eustachian tube, empty into the deep cervical and retropharyngeal glands.

Nerves. The nerves of the auricle and external meatus are derived from the Vth, VIIth, IXth and Xth nerves. The facial nerve sends the posterior auricular branch to the posterior surface of the auricle; the trigeminal, through its third branch in conjunction with some twigs of the auriculo-temporal, supplies the skin of the auricle and external meatus. The cervical plexus also supplies the external ear through its great auricular branch. From the vagus the nerve of Arnold perforates the posterior wall of the meatus and supplies the lining membrane; a larger branch extends along the superior wall of the meatus to the tympanic membrane.

METHODS AND MATERIALS.

The external auditory meatus was removed from one or both sides of all cases coming to autopsy in which permission for examination of the head had been given. The series examined here represents 60 ears, removed from 38 persons. After reflection of the scalp by a post-auricular coronal incision usually used for removal of the calvarium, the cartilaginous and attached membranous portion of the external meatus was excised, carrying the inner end of the section to the tympanum and transecting the canal just within the tragus. Not all specimens could be removed completely, especially at the internal margin, which was sometimes torn away from the bony part of the external acoustic meatus during dissection.

Each specimen was sectioned in four blocks, an outer and inner half of the cartilaginous portion (in adults, this averages 8 mm. in length) and an outer and inner block of the skin and subcutaneous tissue detached from the osseous portion. These blocks were oriented by marking their outer anterior edges with India ink. Sections were cut by the step-section technique, that is, a series of six to ten sections serially with interruptions of about 0.5 mm. In this way the detailed histology of each 0.5 mm. of the external meatus was made available for study, while the total number of sections was held within reasonable limits.

The age grouping of the specimens examined was as follows: Newborn, 12; 1 to 10 years, 4; 10 to 20 years, 2; 20 to 50 years, 9; over 50 years, 11.

Since the objective was the study of the normal, evidence of pathologic processes of the ear eliminated the specimen. Three cases (two in the age bracket 20 to 50 and one over 50) were thus discarded.

OBSERVATIONS.

In the specimens in this series, the lining epithelium of the meatus was as a rule abundantly keratinized, even in infants. The layer of connective tissue just beneath the epidermis has a discrete appearance because of its loose texture and more prominent capillaries.

The glandular elements were found to vary in different parts of the meatus as follows:

	Sebaceous Glands	Apocrine Glands
Outer Cartilaginous	Numerous	Moderate Number
Inner Cartilaginous	Numerous to Few	Numerous
Osseous	Rare	Occasional

The relationship of the apocrine ducts to the adjacent structures was variable. In a count of 100 ducts which could be traced in sections, 83 opened into pilosebaceous units; 12 were traced to direct openings on the surface, and the rest seemed to have branches suggesting the possibility of one gland having both types of exit. Occasionally an apocrine duct showed an expanded segment, a type of ampulla or utricle, lined by stratified epithelium close to the surface.

The epithelium of the apocrine glands varied in height and in content of secretion. Tall columnar cells were found in smaller acini, and low cuboidal ones in dilated acini. Adjacent glands may show this variation as a normal phenomenon. The cells had spherical nuclei basally situated, with pigment granules between them and the free border. On the external border, the epithelium was backed by a layer of elongated cells and a hyaline membrane, which appeared distinctly in all locations and age groups. The degree of acinar dilatation which is found in normal tissue can be judged by the variety shown in Figs. 1 to 7.

In cross-section of the external meatus at all levels, from the outermost to the innermost, blood vessels and nerves were readily observed and formed a pattern which was consistent enough to be described. Sections obtained from the osseous part of the meatus have included only the soft tissue within the bone, while those taken through the cartilaginous portion have demonstrated the vessels and nerves on both the outer and inner surfaces of the cartilage.

The blood vessels were found to be distributed in the different layers of the meatal wall in the following way: In the outermost layer, whether in the membrane within the bony portion or on the surface of the cartilaginous portion, the arteries had subdivided so that three or more groups of arterial vessels were seen at various distances from each other on the circumference of the canal. The arterioles are 20 to 40μ in diameter and the venules somewhat larger and more variable (see Fig. 1).

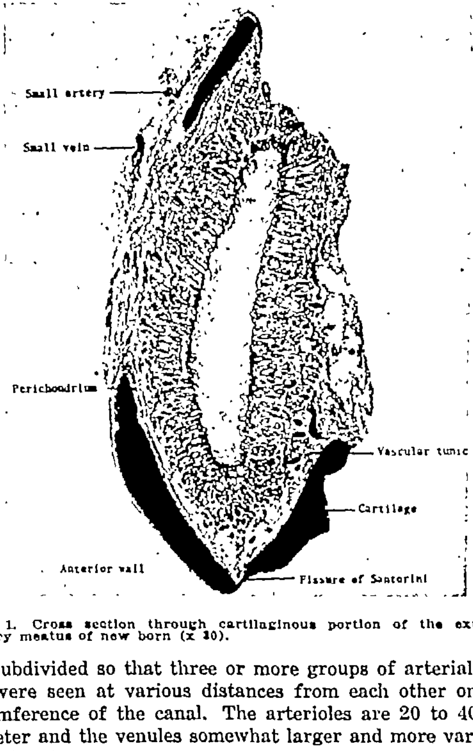

Fig. 1. Cross section through cartilaginous portion of the external auditory meatus of new born (x 30).

Penetration of the blood vessels through the fissures of Santorini and through the membranous layer of the superior and posterior walls of the canal was regularly noted (see Figs. 1, 2). Arborization in a circular pattern occurred in the internal surface of the perichondrium and at a corresponding depth in the osseous portion just outside the deepest of the apocrine (ceruminous) glands. The vascularity of this layer was often striking, and the vessels stood out especially clearly on account of the looseness of the fibrous structure (see Figs. 1, 2). In some specimens where the presence of passive congestion had caused distention of the capillaries, the appearance was almost comparable to the plexiform network of the nasal mucous membrane (see Fig. 2). Thus, this layer is referred to here as the "vascular tunic."

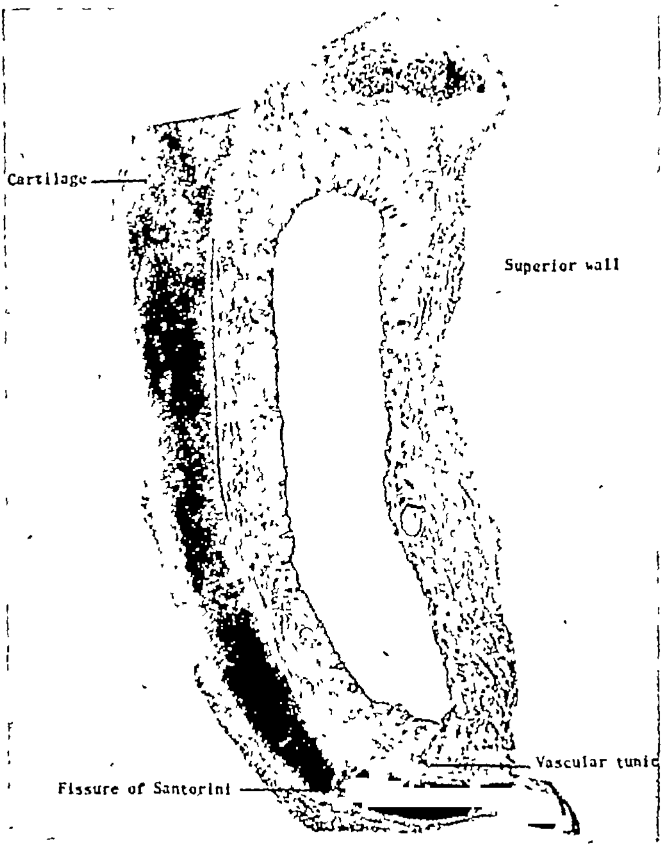

Fig. 2. Cross section through the cartilaginous portion of the external auditory meatus of adult (x 20).

The vascular distribution to the integument is seen to be derived from branches, directed toward the lumen. They are, therefore, at right angles to the predominantly encircling vessels of the vascular tunic (see Fig. 3 DV).

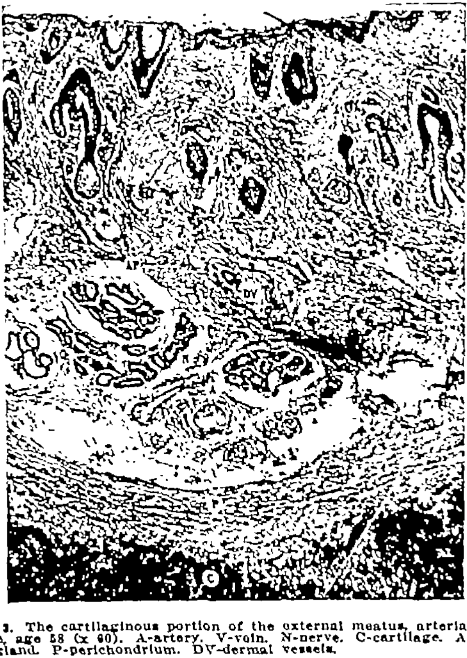

Fig. 3. The cartilaginous portion of the external meatus, arterial wall. Female, age 58 (x 60). A-artery. V-vein. N-nerve. C-cartilage. AP-apocrine gland. P-perichondrium. DV-dermal vessels.

Nerve branches were seen abundantly in the outer layers of the wall of the external meatus in all sections examined (see Figs. 3, 4, 5, 6). The position of the nerve bundles has not been any clue to their identification, except that as a rule the nerves lying anteriorly are probably correctly assumed to be derived from the auriculo-temporal branch of the mandiposterior walls of the canal was regularly noted (see Figs. 1, 2). Arborization in a circular pattern occurred in the internal surface of the perichondrium and at a corresponding depth in the osseous portion just outside the deepest of the apocrine (ceruminous) glands. The vascularity of this layer was often striking, and the vessels stood out especially clearly on account of the looseness of the fibrous structure (see Figs. 1, 2). In some specimens where the presence of passive congestion had caused distention of the capillaries, the appearance was almost comparable to the plexiform network of the nasal mucous membrane (see Fig. 2). Thus, this layer is referred to here as the "vascular tunic."

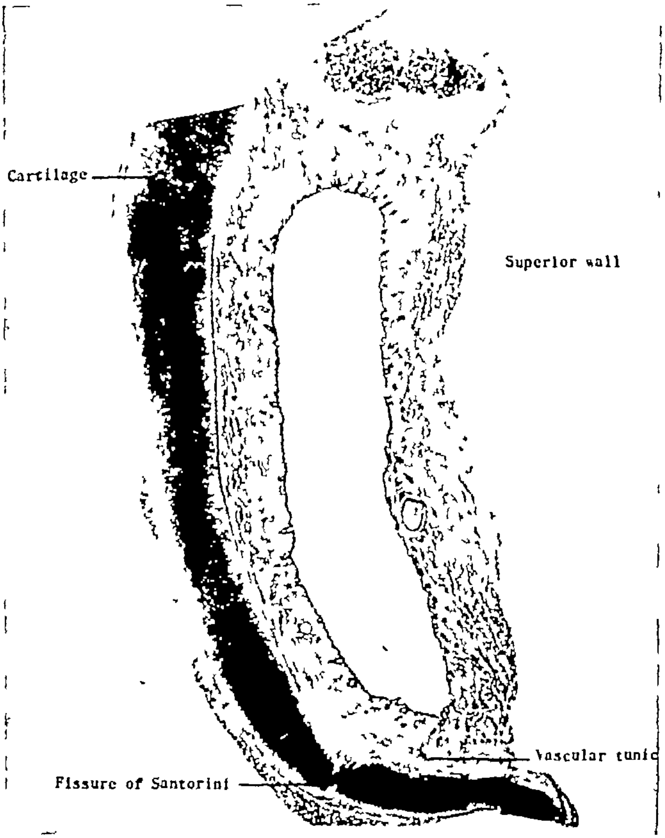

Fig 2 Cross section through the cartilaginous portion of the external auditory meatus of adult (x 20)

The vascular distribution to the integument is seen to be derived from branches, directed toward the lumen. They are, therefore, at right angles to the predominantly encircling vessels of the vascular tunic (see Fig. 3 DV).

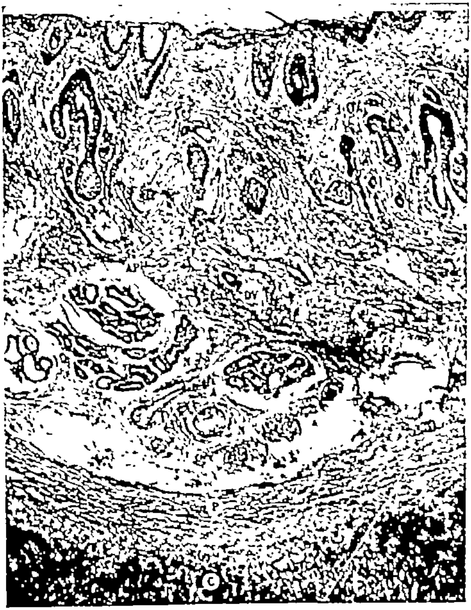

Fig. 3. The cartilaginous portion of the external meatus, arterial wall. Female, age 58 (x 60). A-artery. V-vein. N-nerve. C-cartilage. AP-apocrine gland. P-perichondrium. DV-dermal vessels.

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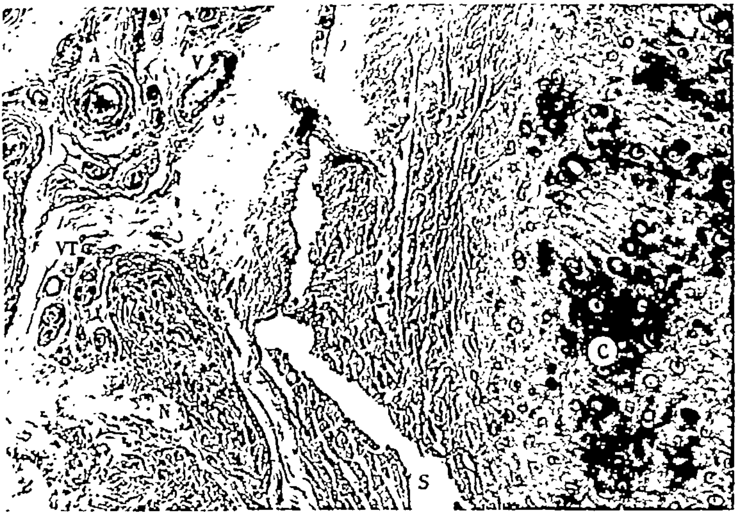

Fig. 4. Cross section at a fissure of Santorini (x 150). S-fissure of Santorini. C-cartilage. N-nerve bundle. A-artery. V-vein. VT-group of small vessels in the vascular tunic.

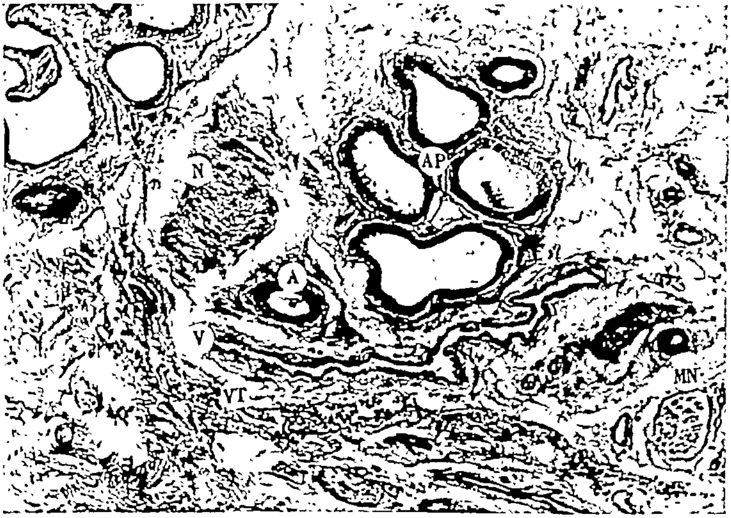

Fig. 5. Structures in relation to the Vascular Tunic (x 150). VT-small vessels of the vascular tunic. A-artery. V-vein. N-nerve. MN-medullated nerve. AP-apocrine gland.

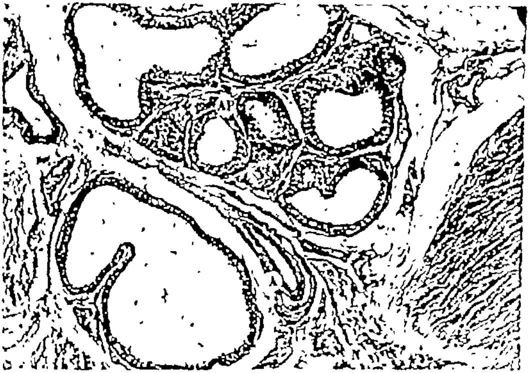

Fig. 6. Vessels and nerves of the glandular layer (x 150). N-nerve (continuous from Fig. 4). A-artery. AP-apocrine glands.

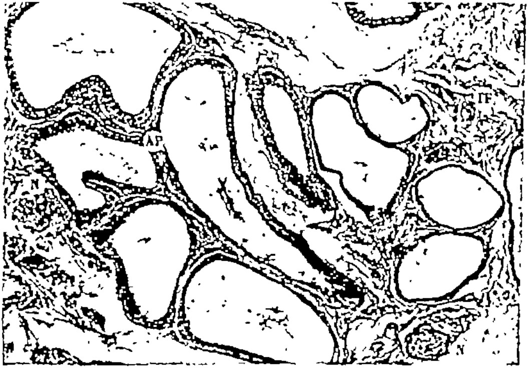

Fig. 7. Nerve endings in skin of external meatus (x 150). AP-apocrine glands. N-nerve. TF-terminal filaments of nerve.

integument of the external auditory canal and to the presence of many nerves. Some of the latter seem to be vasomotor, others secretory. The influence of the structures described in modifying the tissue reactions has been discussed.

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ANNOUNCEMENT.

An introductory course in Reconstructive Surgery of the Septum and External Nasal Pyramid will be given, under the direction of Dr. Maurice Cottle, March 19 through 26, 1955, at the University of Oregon Medical School. This will be sponsored by the Department of Otolaryngology.

REMOVAL OF METALLIC FOREIGN BODY IMBEDDED IN THE RETROPHARYNGEAL SPACE.

Method of More Precise Localization.

LEON M. ARNOLD, M. D.,
and
MAX L. SOM, M. D.,
New York, N. Y.

The localization and removal of small foreign bodies from soft tissues may be an exceedingly difficult and precarious procedure. It is well-known how unpredictable are the results of attempts at operative removal of minute foreign bodies imbedded in the finger or soft tissues, even with wide exposure. The difficulties are much greater when the foreign body is imbedded in the pharynx or esophagus. Here the search is made through a narrow tube at a distance from the eye and in a confined field without benefit of binocular vision. Biplane fluoroscopy is of great help in these cases, but is not available in all hospitals where peroral endoscopy is performed. In its absence, more precise localization of foreign bodies must be attained. A simple method of localization by introducing markers was used in the following unusual case.

J. F., a man 50 years of age, was eating braised beef in a local restaurant. This should not ordinarily contain bones or other hard objects. After swallowing the second mouthful, the patient was stabbed in the throat by a sharp object. Immediately thereafter, he began to choke and tried to induce vomiting by inserting a finger into the throat. Eventually, the choking sensation ceased, and the patient left the restaurant without finishing his meal. He soon noted that he had a stiff neck, and that it became necessary that he hold his head to one side. Any movement of his head or neck produced sharp pain. Later that evening he called his medical doctor who saw no foreign body and prescribed massages and salicylates for his stiff neck.

During the ensuing 24 hours, the patient could neither swallow nor turn his head because of pain. The following day he went to a masseur to have his neck massaged. The dysphagia continued and was accompanied by severe pain in the throat and immobility of the head. This forced him to hold his head in the attitude of one with a severe torticollis.

Forty-eight hours after having eaten the braised beef, the patient presented himself for examination. At this time he had a of 102.6 and appeared to be ill and flushed. He had severe moving the thyroid cartilage and hyoid bone and localized tenderness over the lateral side of the neck. There was fixation

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head and neck due to spasm of the cervical muscles. There was no crepitus or apparent external swelling.

Examination of the pharynx revealed intense edema of the posterior pharyngeal wall with the formation of two longitudinal swellings separated by a midline vertical groove which represented the median raphe There was marked forward displacement of the posterior pharyngeal wall, extending from the hypopharynx up to the level of the uvula. No ulcerations or foreign bodies were seen.

Examination of the larynx revealed marked edema of both arytenoids, which prevented visualization of the aperture of the larynx.

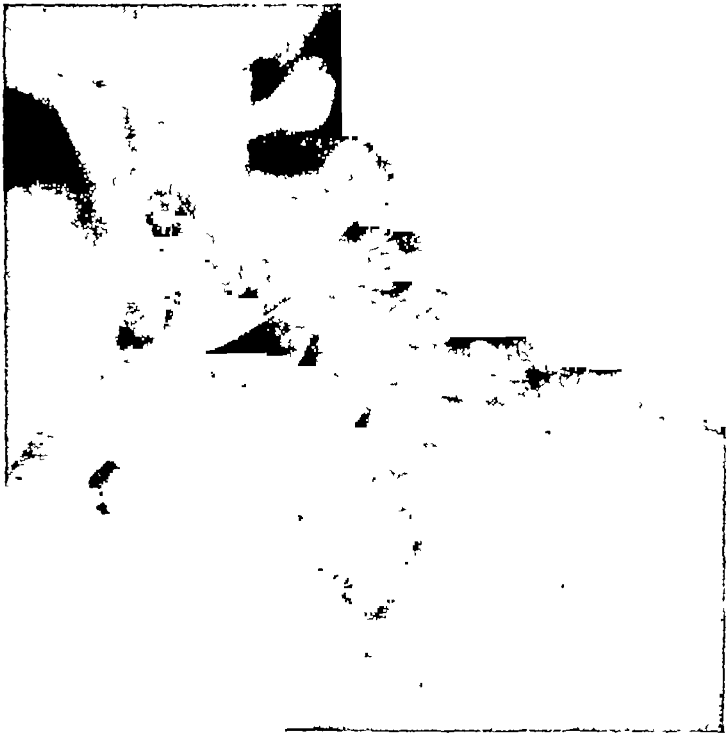

Fig. 1 Lateral radiograph of neck showing wire imbedded in retropharyngeal space. The marked retropharyngeal swelling and accompanying narrowing of the airway is seen No free air is visible in the soft tissues.

The patient was admitted to the Beth Israel Hospital on November 7, 1950, where X-rays showed a metallic wire-like foreign body 1½ inches long and 1 mm. in diameter lying in the retropharyngeal tissues at the level of the third cervical vertebra (see Fig. 1). This foreign body lay transversely and extended across the midline. There was marked widening of the retropharyngeal space with accompanying narrowing of the pharyngeal airway. The wire was obliquely situated, the right end being somewhat lower than the left and nearer the vertebrae. There was no free air in the retropharyngeal space (see Fig. 2).

The patient was given large doses of parenteral chemotherapy in the form of Penicillin G, fortified by procaine penicillin, in conjunction with streptomycin. The next morning the patient's larynx was thoroughly anesthetized with cocaine and adrenalin, and a careful search was made for the foreign body. By means of a closed curved forceps, all folds of the pharynx were carefully probed. In addition, with the aid of direct hypopharyngoscopy, the pharynx was explored for a possible clue as to the entering point of the foreign body. This was entirely unsuccessful.

The following day, November 9, four days after ingestion of the foreign body, the patient was again premedicated with demerol and atropine. His pharynx was cocainized, and intravenous sodium pentothal and curare were administered as a general anesthesia. By suspension laryngoscopy, using a Lynch apparatus, the head was suspended, thus exposing the posterior hypopharyngeal wall down to the post-cricoid region.

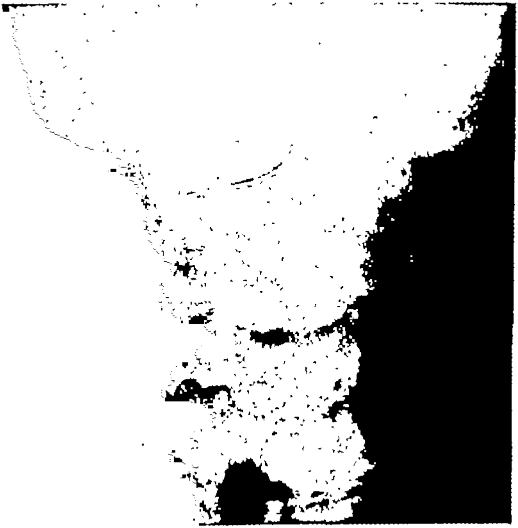

Fig. 2. Antero-posterior view of neck showing wire in transverse position at level of third cervical vertebra.

A longitudinal incision was made to the right of the midline, starting from a point opposite the lower pole of the tonsil and terminating at the level of the arytenoids. It was felt that the knife could encounter the long axis of the foreign body, and would thereby reveal its location.

The incision was deepened down to the anterior surface of the vertebrae. An intensive search was made, but no foreign body could be seen or palpated, and the procedure was abandoned.

A small amount of slough was seen in the deep tissues of the retropharyngeal space, but no actual pus Because of the extensive retropharyngeal swelling and the additional operative trauma, it was deemed safer to perform a tracheotomy

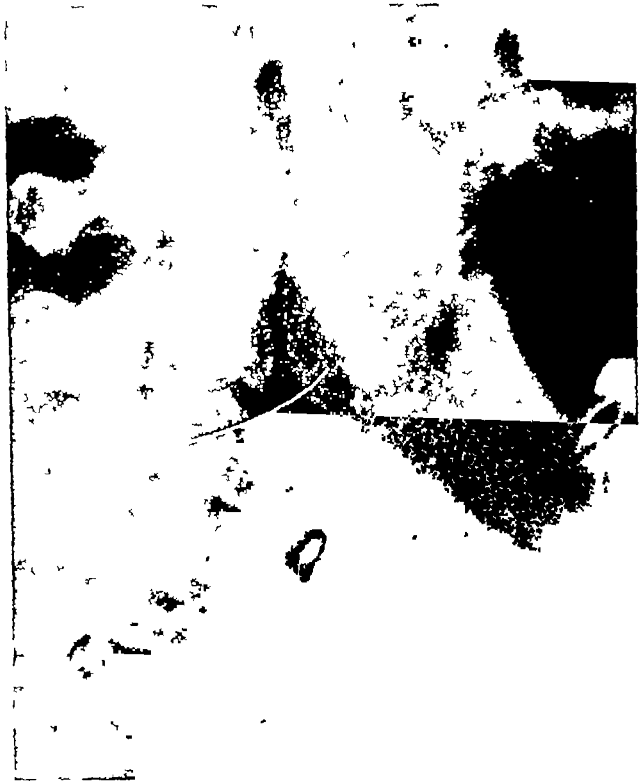

Fig 3 Lateral view of neck showing markers The foreign body lies between the steel wire suture above and the Michel clip below

Intensive biotherapy with penicillin and streptomycin was continued. The patient's temperature remained elevated and retropharyngeal swelling persisted His nutrition was maintained by means of intravenous fluids and vitamins

Five days after the first attempt at removal, the patient was returned to the operating room. External operation was considered, but the difficulties to be encountered were deemed just as great as with the transoral route. The pharynx was cocainized and general anesthesia administered.

A Berman foreign body locator was unsuccessfully employed in attempting to locate the foreign body. This instrument produces a whistling sound which increases in intensity when it approaches a metallic object. This foreign body was apparently too fine to register on the machine.

At this point it became apparent that more precise localization of the foreign body was essential. Since the approximate location of the foreign body was known in relationship to the vertebrae, it was decided to localize the wire more definitely by fixing a radio-opaque object to the mucosa above and below the approximate level of the foreign body. This relationship could then be confirmed by further Roentgen films. A steel wire suture was placed in the area, which we estimated to be the uppermost level of the foreign body, and a Michel clip at the lower level. Radiographs revealed the foreign body to be between the wire and the Michel clip. These relationships would remain fixed in any position of the head. A similar method has been employed in locating fragments of needles buried in the fingers. A suspension laryngoscopy was again performed and adequate exposure of the hypopharynx obtained.

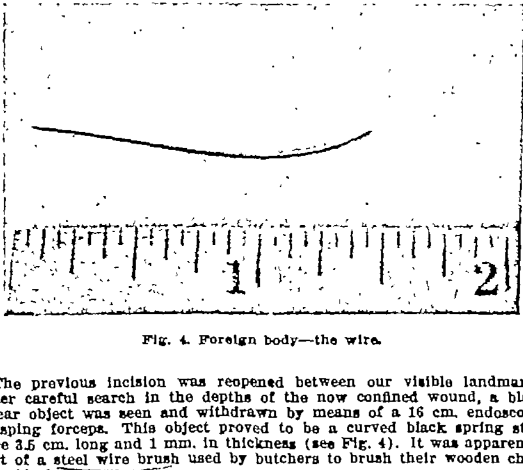

Fig. 4. Foreign body—the wire.

The previous incision was reopened between our visible landmarks. After careful search in the depths of the now confined wound, a black linear object was seen and withdrawn by means of a 16 cm. endoscopic grasping forceps. This object proved to be a curved black spring steel wire 3.5 cm. long and 1 mm. in thickness (see Fig. 4). It was apparently part of a steel wire brush used by butchers to brush their wooden chopping blocks.

The bio-therapy was continued, and after a week the edema had sufficiently subsided to permit removal of the tracheotomy tube. The patient began to take fluids and food by mouth, and was discharged three weeks after entering the hospital with the wound in the posterior pharyngeal wall granulating well. Complete healing occurred after another two weeks. The patient continued to have some stiffness in the neck and pain in moving his head and neck. He has now completely recovered.

SUMMARY.

A case of a fine metallic foreign body imbedded in the retropharyngeal space, which posed real difficulties in removal, has been presented. The significant features in the management of this case are:

1. The use of the Lynch suspension apparatus proved invaluable. Removal by the usual direct laryngoscopy would have been extremely difficult, if at all feasible, without the excellent exposure permitted by suspension laryngoscopy and without the freedom of manipulation by two hands which this method permits.

2. The use of metallic markers to restrict the field of search saved needless probing into edematous tissue in seeking the foreign body. The usual bony landmarks, such as the hyoid bone and thyroid cartilage, which help in roentgenographic localization, are not helpful at the time of endoscopy. The radiographic localization in terms of the vertebrae, or other fixed landmarks, is also not sufficient, because in hyperextending the head and neck in the laryngoscopy, these relationships are distorted. On the other hand the relationships of the foreign body to visible objects temporarily fixed in the mucous membrane, after this relationship has been demonstrated radiologically, is a constant reminder of the field of search.

3. The Berman foreign body locator is of value in localizing larger metallic foreign bodies. It proved to be of no value in localizing the very thin wire in this case. Had the biplane fluoroscope been available, it is likely that less difficulty would have been encountered in localizing the foreign body.

A REDESIGNED DIRECTOR FOR USE IN RHINOPLASTY.*

ALBERT P. SELTZER, M.D.,
Philadelphia, Pa.

One of the tests of skill for the surgeon operating on the nose has often been the exactness with which the cartilages could be cut through. These tissues are particularly elastic in the nose, and so, like rubber, they are difficult to cut through in a straight line and a clean incision.

To avoid the undesirable results that are oftentimes met, a new type of director has been devised for use, particularly in dividing the lower lateral cartilages, when operating on the nasal tip.

The instrument customarily used has been the Neivert director,[1] which is a narrow strip of metal, curved sharply forward in a hook, at the distal end. From this hooked extremity there is a narrow, medial groove extending backward, which is designed to be followed by the knife when making the incision. One drawback in this design is that the director is apt to be put in motion by the action of the knife's cutting through the cartilage (see Fig. 1-a).

The writer's design, (see Fig. 1-b) is a clamp of stainless steel, in the type of the hemostat. The clamp is six and three quarters inches (17 cm.) in overall length, with one blade a quarter of an inch (6 mm.) longer than the other. On the longer blade, the excess length is hooked in a parrot-like beak, at an obtuse angle toward the shorter. Both blades are cross dull-ridged throughout their entire length. They are also fenestrated for nearly the entire length, the slit being wide enough to admit a Bard-Parker No. 15 knife blade.

To use the clamp in operating, the cartilage is drawn out-

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* From the Ear, Nose and Throat Department, Graduate Hospital, University of Pennsylvania.

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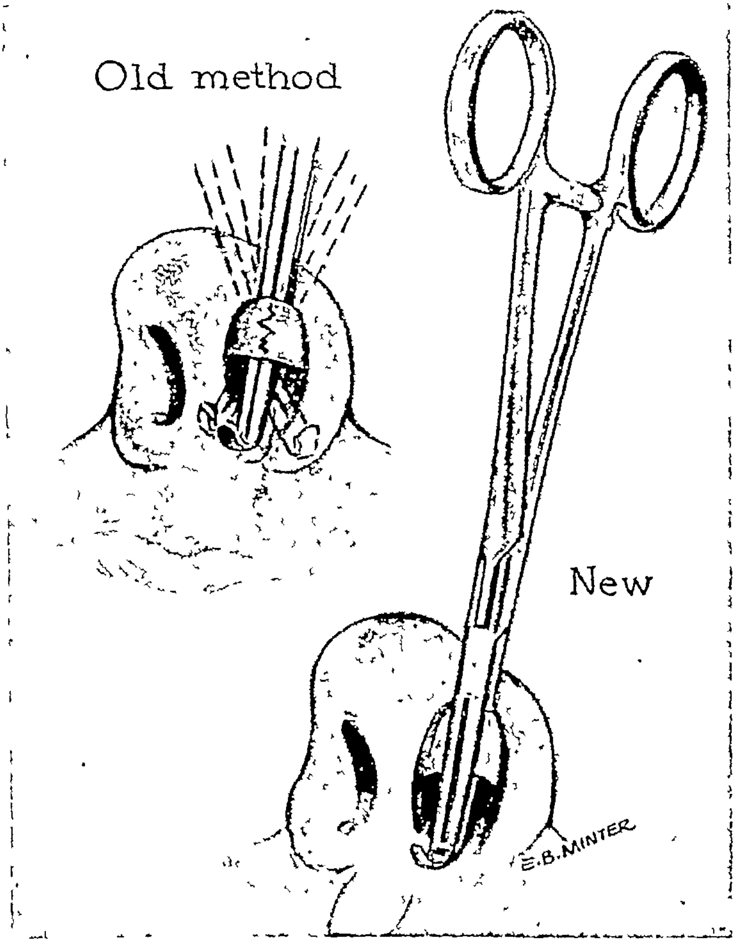

Fig. 1 (a) Nelvert director in use; (b) New design for comparison.

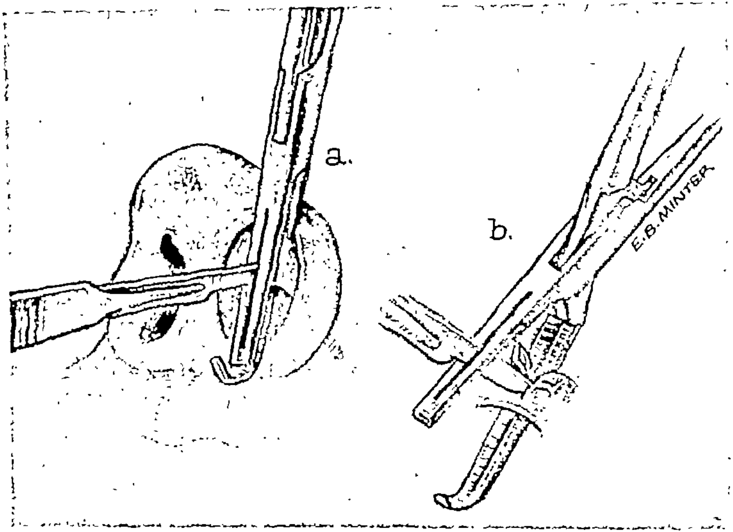

Fig. 2. (a) Seltzer clamp, with knife in position; (b) Clamp blades opened showing knife cutting through and through cartilage.

side the nostril through the primary incision, and so applied that the blades grasp the cartilage, with the slit in the blades following exactly the line of the proposed incision. This may be through the angle of the cartilage, or through any chosen location. By this means, both cartilages can be cut in exactly the same way and with the same degree of accuracy. The final result is that the tip of the nose will be entirely symmetrical, with the external nasal openings appearing exactly alike.

REFERENCE.

1. SELTZER, A. P.: Plastic Surgery of the Nose. Phila. J. B. Lippincott Co., p. 143, 1949.

Note: The writer wishes to thank George F. Pilling & Son for making this instrument according to the specifications.

2104 Spruce Street.

THE ASSOCIATION OF MILITARY SURGEONS OF THE UNITED STATES.

THE MILITARY SURGEON.

ANNOUNCEMENT.

The 61st Annual Convention of the Association of Military Surgeons of the United States will be held November 29 to December 1, 1954, at the Hotel Statler, Washington, D. C. The program will cover a wide range of professional and scientific subjects plus the latest advances in medical and allied sciences, the latter to be shown in a large variety of technical exhibits and films.

For the entertainment of the members and guests, a full schedule of events is being arranged. As in the past, one of the outstanding features is the Honors Night Dinner on December 1, at which the Sir Henry Wellcome medal and prize, the Gorgas medal, the Stitt award, the McLester award, the Louis Livingston Seaman prize, and the Founder's medal will be presented.

The granting of credit points for retirement to eligible Reserve Officers for attendance at meetings is expected to be announced soon by the Department of Defense.

IRRIGATION OF NASOPHARYNX AFTER ADENOIDECTOMY.

D. A. DeLisa, M.D.,
Schenectady, New York.

I have had no postoperative hemorrhage following adenoidectomy since I began irrigating the nasopharynx with cold saline at the conclusion of the operation. This technique is reported with the hope that it will prove as helpful to others as it has to me.

At the end of the operation, after the gauze pack is removed from the nasopharynx, saline at about 60° F. is flushed into each nostril with a rubber bulb syringe. The suction tip is held in the pharynx at the same time, and the saline is aspirated before it can reach the glottis. The irrigation is repeated until complete hemostasis is obtained. All clots are washed out, and the vessels are constricted by the cold saline. Incidentally, the patient has a clear nasal airway and is more comfortable after operation without clots in the nasopharynx.

Direct inspection of the nasopharynx, using a headlight and palate retractor, confirm complete removal of tissue and hemostasis. Incomplete removal of tissue is the commonest cause of postoperative hemorrhage. There is no substitute for seeing with the eyes that all tissue and clots are removed.

I do not believe that the irrigation technique will completely eliminate postoperative hemorrhage, but in the 493 cases in which I have used it, not one has occurred. Previously my incidence was about 3 per cent. Incidentally, no medication intended to prevent bleeding is prescribed, but aspirin is prohibited.

1408 Union Street.

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication July 10, 1954.

THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC., MEETINGS.

MID-WINTER.

Eastern Section Meeting..........Friday, January 7, 1955
Philadelphia, Pa., Hotel Warwick

Triological Council Meeting..........Saturday, January 8, 1955
New York City, The Waldorf-Astoria

Middle Section Meeting..........Monday, January 14, 1955
Detroit, Mich., Sheraton-Cadillac Hotel

Western Section Meeting..Saturday-Sunday, Jan. 15-16, 1955
Los Angeles, Cal., The Town House

Southern Section Meeting..........Saturday, January 22, 1955
Charlottesville, Va., Medical School Auditorium

SPRING.

American Board of Otolaryngology
Richmond, Va., March 6-10, 1955

All Society Meetings will be held at the Hollywood Beach Hotel, Hollywood, Florida.

American Laryngological Association..........March 13, 14, 1955

Triological Society.....(mornings only) March 15, 16, 17, 1955

American Broncho-Esophagological Association
(afternoons only) March 15, 16, 1955

American Otological Society, Inc..........March 17, 18, 1955

The Hollywood Beach Hotel (American plan) is a delightful place to hold the Spring Meetings — you may wish to bring the family along. The Hotel offers its guests such features as ocean bathing, deep sea fishing, 18-hole golf course and country club, tennis and many other activities.

For hotel reservations please communicate with Mr. John W. Tyler, Manager, Hollywood Beach Hotel, Hollywood, Florida.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

September 1, 1954.

Acousticon Models A-17, A-180 and A-185.
Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 1, New York.

Auditone Models 11 and 15.
Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67 and 70.
Manufacturer: Audivox, Inc. 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.
Manufacturer: Aurex Corp. 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac Model M; Mono-Pac Model "Lyric"; Mono-Pac Model "Rhapsody."
Manufacturer: Beltone Hearing Aid Co., 2900 West 36th St., Chicago 32, Ill.

Cleartone Model 700.
Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Junior Model D-2; Dahlberg Model D-3 Tru-Sonic; Dahlberg Model D-4 Tru-Sonic.
Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Fortiphone Models 19-LR; 20A; 21-C and 22.
Manufacturer: Fortiphone Limited, Fortiphone House, 247 Regent St., London W. 1, England.
Distributor. Anton Hellman, 75 Madison Ave., New York 16, N. Y.

Gem Hearing Aid Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.
Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.
Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico Model J; Maico Top Secret Model L; Maico Maxitone.
Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis, Minn.

Micronic Model "Mercury."
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Model T-10; Microtone Model T-612.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

Normatone Model C and Model D-53.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.

Distributor: Normatone Hearing Aid Co., 22 East 7th St., St. Paul (1), Minn.

Otarion Models B-15 and B-30; Otarion Models F-1, and F-3; Otarion Model H-1; Custom "5."

Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40. Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny Myte).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Model 62 Starlet; Model 72; Model 82 (Zephyr).

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Distributor: Radioear Corp., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Silvertone Model H-16, J-92; Silvertone Model P-15.

Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.

Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966; Sonotone Model 977; Sonotone Model 988.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Televox Model E.

Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 99; Telex Model 200; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 953; Telex Model 1700.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.

Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster; Model Cameo.

Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill

Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2.

Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal; Zenith "Regent."

Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices have vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TRANSISTOR HEARING AIDS ACCEPTED.

Acousticon Model A300; 1 transistor, 2 tubes. Model A-310; 1 transistor; 2 tubes. Model A-330; 3 transistors. Model A-335; 3 transistors.

Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 35, New York.

Audivox, Model 71; 3 transistors.

Manufacturer: Audivox, Inc., 123 Worcester St., Boston 18, Mass.

Beltone Concerto Model; 3 transistors.

Manufacturer. Beltone Hearing Aid Co., 2900 W. 36th St., Chicago 32, Illinois.

Maico Transist-Ear, Model O; 3 transistors.

Manufacturer: The Maico Company, Inc., 21 N. 3rd St., Minneapolis 1, Minnesota.

Micronic "All American" Hearing Aid; 3 transistors.

Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Model T1 (Red Dot); 3 transistors. Microtone Model T1 (Yellow Dot); 3 transistors. Microtone Model T31 (Micro-Mite); 1 transistor and 2 tubes. Microtone Model Skylark; 3 transistors.

Manufacturer: The Microtone Corporation, Ford Parkway on the Mississippi, St. Paul 1, Minn.

Otarion Model C-15; 1 transistor, 2 tubes. Otarion Model D-1; 3 transistors. Otarion Model F-22; 1 transistor, 2 tubes.

Manufacturer: Otarion, Inc., 4757 N. Ravenswood Ave., Chicago 40, Ill.

Paravox Model K.M. (TRANSonic) ; 3 transistors.

Manufacturer: Paravox, Inc., 2056 East 54th St., Cleveland, Ohio.

Radioear Model 820; 3 transistors.

Manufacturer: E. A. Myers & Sons, Inc., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Silvertone Model H-25; 3 transistors.

Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.
Distributor: Sears, Roebuck & Co., 925 South Homan Ave., Chicago 7, Illinois.

Sonotone Model 1010; 1 transistor, 2 tubes. Model No. 1111; 3 transistors.

Manufacturer: Sonotone Corporation, Elmsford, N. Y.

Telex Model 954; 1 transistor, 2 tubes. Telex Model 956; 3 transistors.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Unex Model TR-3D; 3 transistors.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Zenith Model Royal-T; 3 transistors. Zenith Model Super Royal-T; 3 transistors.

Manufacturer: Zenith Radio Corp., 5801 W. Dickens Ave., Chicago 39, Illinois.

SEMI PORTABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).

Manufacturer: A. M. Brooks Co., 1222 W. Washington Blvd., Los Angeles 7, Calif.

Aurex Hearing Aids (three types).

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.

Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. D. E. Staunton Wishart, 170 St. George St., Toronto 5, Ontario, Canada.
Vice-President: Dr. Wm. J. McNally, 1509 Sherbrooke St., West Montreal 25, Canada.
Secretary-Treasurer: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Illinois.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Hollywood Hotel, Hollywood, Fla., March 17-18, 1955.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. Henry B. Orton, Newark, N. J.
First Vice-President: Dr. Jas. H. Maxwell, Ann Arbor, Mich.
Second-Vice-President: Dr. Clyde A. Heatley, Rochester, N. Y.
Secretary: Harry P. Schenck, Philadelphia, Pa.
Treasurer: Fred W. Dixon, Cleveland, Ohio.
Librarian-Historian: Dr. Bernard J. McMahon, St. Louis, Mo.
Meeting: Hollywood Hotel, Hollywood, Fla., March 13-14, 1955.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Kenneth M. Day, 121 University Pl., Pittsburgh, Pa.
President-Elect. Dr. Dean M. Lierle, Iowa City, Iowa.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Eastern Section Meeting, Friday, January 7, 1955, Philadelphia, Pa., Hotel Warwick.
Triological Council Meeting, Saturday, January 8, 1955, New York City, The Waldorf-Astoria.
Middle Section Meeting, Monday, January 14, 1955, Detroit, Mich., Sheraton-Cadillac Hotel.
Western Section Meeting, Saturday - Sunday, January 15 - 16, 1955, Los Angeles, Cal., The Town House.
Southern Section Meeting, Saturday, January 22, 1955, Charlottesville, Va., Medical School Auditorium.
Meeting: Hollywood Hotel, Hollywood, Fla., March 15-16-17, 1955, morning only.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

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Time and Place: 1956, Puerto Rico.

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Meeting: Waldorf-Astoria, New York City, September, 1954.

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Meeting: Fifth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
President: Dr. J. H. Font, Medical Arts Bldg., San Juan, P. R.
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Meeting: 3rd Int [illegible] ational Congress of Broncho-Esophagology.
Time and Place: [illegible] tember or October, 1954, Lisbon, Portugal.

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General Secretary: Dr. Paul Holinger, 700 No. Michigan Ave., Chicago (11), Ill.
Meeting: Statler Hotel, Washington, D. C., May 5 - 10, 1957.

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Sede: Avenida da Liberdade, 65, 1.°, Lisboa.

THE
LARYNGOSCOPE.

VOL. LXIV OCTOBER, 1954. No. 10

THE COMBINED USE OF ANTIBIOTIC INSUFFLATION AND VASODILATOR THERAPY IN OZENA.*†

H. J. STERNSTEIN, M.D.,
Boston, Mass.

INTRODUCTION.

Since ozena was first described by Fraenkel[1] in 1876, as a distinct clinical entity, numerous attempts have been made to establish a known etiology as a rational basis for treatment. Failure to solve the problem thus far has led to the growing opinion that the disease is not due to any one single cause but very likely is the product of a combination of factors.[2,3] In view of this trend, the significant though limited results obtained with recent independently formulated methods of streptomycin[4-8] and vasodilator[9-14] therapy suggested a combined approach for clinical investigation. In 1951, I reported[15] a new method for the treatment of ozena by combining the *local use of aqueous streptomycin and other antibiotics with* vasodilators in oral form. Histamine, priscoline,[16-17] and niacin were employed to stimulate more profound local effects on the vascular and neurotrophic changes through a potential increase in the peripheral circulation of the nose by their combined parasympathetic and sympatholytic actions. The preliminary results suggested that properly selected antibiotics and the multiple use of vasodilators could effectively eliminate odor and crusts and equally tend to promote a reversal of the regressive changes in this disease.

A diagnosis of ozena as differentiated from the secondary form of atrophic rhinitis was based on the following criteria: odor, green crust formation, atrophy and gram negative or-

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* Presented as a Candidate's Thesis to the American Laryngological, Rhinological, and Otological Society, Inc., July, 1953. (Revised and condensed from the original manuscript).

† From the Department of Otolaryngology, Massachusetts Eye and Ear Infirmary.

Editor's Note: This ms. received in the Laryngoscope Office and accepted for publication, May 11, 1954.

ganisms consisting of either Friedländer's bacillus, B. coli, B. pyocyaneus or B. proteus.

The purpose of this paper is to present a modification of the earlier use of aqueous antibiotics to the more recent method of insufflation with micronized streptomycin, Terramycin, and chloramphenicol (chloromycetin). The data, results, and follow-up studies in a total series of 35 cases observed over a period of three years are reported including three case reports with X-ray, and microscopic evidence of turbinal growth and regeneration of respiratory epithelium.

LITERATURE.

Etiology: In 1896, when K. ozena was isolated by Abel, it was believed that a bacterial origin of the disease had been discovered.[18] Later investigators found no consistency in the bacterial flora, except possibly for the close morphologically related K. pneumoniae (Friedländer's bacillus)[2,20,22] which proved to be nontransmissible.[19] Further evidence that klebsiella are principally secondary invaders has been suggested.[24a,4,5,27] More recently C. diphtheriae (mitis type) was offered as the primary cause.[42]

Pathogenesis: Pollock (1931) believed that either primary[23] or secondary[24] sphenopalatine ganglion degeneration was induced by preceding sinusitis, resulting in atrophic changes of the nasal mucosa and finally ozena. Heredity, abnormal structural patency, scarlet fever, and diphtheria were mentioned as predisposing and exciting factors. Ballenger[24a] observed, however, that the true cause of ganglion atrophy was still unexplained. Endocrine deficiency and the lack of hormonal secretion was advanced as a principal cause of atrophy by Dorochenko[26] (1937), Mortimer et al[27] (1937), Blaisdell[25] (1938), and Ruskin [28,28a] (1942). Other factors considered by Taptas[29] were toxic destruction of nasal mucosa either primary or secondary to a slow progressive diphtheria of low virulence, and saphrophytic agents resulting from the stagnation of secretion.

Histopathology: No agreement exists as to the uniformity of the particular epithelial and vascular changes inasmuch as adequate histological studies of the so-called normal turbinates are lacking. The common pathological findings[2,8,10,20,20a] may be summarized as follows: *a*, Epithelium: metaplasia from ciliated columnar to stratified squamous, with the respiratory mucous membrane still retained in a few protected areas: *b*, Basement membrane: normal to thickened or complete disappearance, *c*, Tunica propria: lymphocytic and plasmocytic infiltration especially concentrated around the mucous glands: minimal to advanced perivascular fibrosis; occasional Russell bodies; deeper vessels thickened, hyalinized and occasionally obliterated; variable fibrosis to atrophy of mucous glands, *d*, Bone: progressive lacunar resorption of the turbinates.

Hormone Therapy: In 1937 and 1988, the local use of estrogenic hormones was reported by Dorochenko,[26] Mortimer, et al,[27], Blaisdell,[25] and later others[31,32] with limited results. Biopsy studies[30] failed to reveal definite changes in a single direction.

Antibiotic Therapy: The chemical effectiveness of streptomycin with the Friedländer organism in mice was first successfully reported by Heilman[33] (1945). Clinical studies by Thornell[3,4] and Simonton[5] later showed a disappearance of odor and klebsiella organisms although a less appreciable effect on crusting. Atrophy remained uneffected. Jannoulis[34] reported only a disappearance of odor with combined penicillin and streptomycin.

Vasodilator Therapy: Various investigators reported encouraging results with histamine ionization[10] and acetylcholine[24,24b,9] attributable to an increased blood supply to the nasal mucosa. Others[12,13] claimed equally good results with vitamin PP and *nicotinic acid*[14] *on* the assumption that ozena was a dysvitaminosis rather than a vasomotor disorder. Procaine block of the stellate ganglion[35] was also reported to diminish headache and fetor, with increased turbinal vasodilation, as further evidence of a vasomotor neurotrophic disturbance.

Surgical Therapy: More recently various surgical procedures for narrowing the bony nasal vault with osteotomy,[30] submucosal implants of plastic[39,40] or bone grafts[43] have been favorably reported. Placental tissue implants showed poor results.[37]

CLINICAL MATERIALS

Ten of 35 patients selected for study were follow-up cases from the original group of 13 first reported.[15] The remaining 25 patients have since been newly added. In the total series, six were men and 29 were women ranging in age from 10 to 73 years, with a duration of symptoms from five to 50 years. Eight patients were treated previously in the outpatient department for a period of about 15 years. Nineteen new patients were referred by the clinic and eight were treated privately since the beginning of this work. Estrogenic nasal sprays, (Estrone 1-10,000 to 1-20,000) had been previously used by eight patients over a period of 10 years and in eight cases sinus surgery was followed by estrogen nasal therapy. Serum tests in all cases were negative.

The clinical findings showed 20 patients with marked odor, slight in two and absent in 13. Although no odor could be detected objectively in eight of the 13 patients on forced expiration, it was nevertheless noticeable on the crusts removed by cotton applicator. Crust formation was generally distributed over the turbinates, septum, nasopharynx and posterior surface of the soft palate. It was marked in 27 cases, moderate to slight in seven, and absent in one. Advanced atrophy (3+ to 4+) was seen in 26 patients, less than (1+ to 2+) in seven, and no gross atrophy in two. Sixteen patients gave a history of complete anosmia, seven partial and 12 no loss of the sense of smell. Nasal blockage and discharge were more or less present. No correlation was observed between the degree of odor, crusting and atrophy.

The bacteriological findings obtained from 35 individual cultures showed a variety of gram negative and gram positive organisms with B. Friedländer's (45%), H. staphylococcus aureus (37%), B. coli (37%), B. proteus (20%) and pneumococcus (11%).

X-rays of the sinuses were reported in 50 per cent of the patients as showing inactive pathological changes. The findings were variable consisting of slight to markedly thickened membrane, a few with added decalcification of the ethmoids and some with either uni- or bilateral sclerotic bone changes. One patient revealed cystic membrane of the antrum and one, three small ivory transplants in the septum. Only 28 per cent indicated active suppuration and thickened membranes. Of this group, one-half showed pus in the antrum, one in the frontal sinus with sclerosed bone and four retained secretion in the ethmoids and antrum with marked bone sclerosis in one. The remaining 22 per cent were negative. Marked to moderate atrophy of the middle and inferior turbinates was demonstrated in all of the cases.

BIOPSY STUDIES.

Biopsies were obtained from the under surface of the inferior turbinate in its approximate middle third section including the lateral and medial aspects. The epithelial changes were found to be variable ranging from columnar to transitional and to stratified squamous epithelium. Cilia and goblet cells were absent except for the occasional suggestion of the former in protected areas on the lateral surface. The apparent lack of vascularity noted clinically was at variance with the histological findings for in some of the more advanced cases of atrophy, larger blood vessels were identified in the deeper portions of the tunica propria despite the marked fibrosis and relatively scant number of capillaries in the subepithelial region. No definite correlation was observed between the microscopic picture and the gross clinical findings.

Comment: Mueller[41] points out that a diagnosis of ozena cannot as a rule be made on the basis of a small biopsy alone without due regard to the clinical picture, as for example in the case of less advanced atrophy (1+ to 2+), where perivascular infiltration and fibrosis may well be within normal limits providing the specimen included the deepest layers down to the periosteum of the turbinate. He believes that all so-called "normal" nasal mucous membrane shows chronic inflammation to a greater or lesser degree, *i.e.*, infiltration with lymphocytes and plasmocytes.

METHOD.

Cultures were taken before and during treatment at approximately monthly intervals. Antibiotic therapy was carried out with the insufflation of either Streptomycin (calcium chloride complex[*]), Terramycin hydrochloride,[†] or Chloramphenicol (Chloromycetin[‡]) in an effort to obtain concentrated effective local administration and dosage. Streptomycin[†] and Terramycin[†] were micronized (10 microns, average size) to facilitate their better distribution throughout the nose and nasopharynx. The micronized antibiotics were mixed with sodium lauryl sulfate, a surfactant, diluted with beta lactose powder and dispensed in capsules of two strengths[§]: the weaker, containing 25 mgs. of antibiotic, 2 mgs. of sodium lauryl sulfate and 70.5 mgs. of beta lactose; the stronger, 50 mgs. of the antibiotic, 2 mgs. of sodium lauryl sulfate and 33 mgs. of beta lactose. These were prescribed in an average daily dosage ranging from 100 mgs. to 200 mgs. Instructions were given to insert the capsule (size No. 5) in the detachable tip of a De Vilbiss No. 119 powder blower either with the extreme ends of the capsule snipped off, or its contents emptied into the same receptacle. The patient was directed to inhale the powder during insufflation. Stinging sensations and lacrimation were at times encountered. If these side effects were not tolerated, aqueous antibiotics were prescribed instead, using bacitracin (1500-2000 u/cc.) or Streptomycin (25,000 u/cc.)[§§] or both.

The use of a particular antibiotic was indicated by the predominant flora and clinical response. With mixed organisms, either streptomycin or one of the broad spectrum antibiotics was employed. When the gram negative organisms became inhibited, further antibiotic therapy was continued with either Terramycin or bacitracin depending upon the culture report, the degree of crusting, and discharge. Gantrisin was prescribed for B. proteus organisms in 25 per cent to 30 per cent aqueous concentrations locally and orally in tablet form (total daily dose 4-6 grams). Sensitivity tests were conducted with concentrations of antibiotics as high as 10 mgs. per cc. of culture fluid. (The maximum level required for systemic concentrations ranges from 0.6 to 256 micrograms per cc. of culture fluid).[15]

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[*] Supplied by Merck & Co., Inc., Rahway, New Jersey.

[†] Supplied by Chas. Pfizer & Co., Inc., Brooklyn 6, New York.

[‡] Chloramphenicol (Chloromycetin) was not available in micronized form for investigation until after this paper was written. Further studies are now in progress with the new material supplied by Parke, Davis & Co.

[§] Prepared at the Massachusetts College of Pharmacy under the supervision of Dr. Leslie M. Ohmart Professor of Pharmacology.

[§§] Streptomycin 1 mg. is equivalent to 1000 μ

Vasodilator therapy was based largely upon the combined use of Priscoline and niacin. Priscoline,*[16,17] was prescribed orally in dosage of 50 mgs. three to four times a day after *meals. Patients were informed of the various side reactions to* expect such as flushing of the face and upper trunk, tingling of scalp, chilliness or warmth of the extremities, increased palpitation, muscle twitchings, general aches and pains in the extremities, epigastric discomfort or abdominal cramps with increased peristalsis. Blood pressure was periodically measured. If signs of postural hypotension such as a fall in blood pressure, low back pains or general weakness appeared, the drug was temporarily discontinued. It was not employed in suspected cases of gastric ulcer or cardiovascular disease. Niacin was prescribed in doses of 50 mgs. from four to eight tablets daily after meals. No side effects were encountered with this drug.

In the management of nasal hygiene, all patients were instructed to discontinue nasal irrigations. Lamb's wool (sterile) in the form of small plugs loosely inserted in each nostril with sufficient room left for nasal breathing was prescribed several times daily for one-half hour intervals to enhance nasal moisture. Caroid (papaine)† was employed in either powder or liquid form, as a modifying solvent of viscid nasal secretions. The optimum concentration was a six per cent (6%) mixture with a pH of 5.4 in a beta lactose powder base. The powder form was administered through the detachable No. 119 De Vilbiss tip, insufflating one-quarter scoopful first upwards and repeated downwards for each nostril. Beta lactose was chosen to promote a lower pH of the nasal secretion for the inhibition of bacterial activity.[18] The liquid form, five per cent (5%) with a pH of 5.0+ was prepared as follows: papaine 5 per cent, beta lactose 6 per cent, glycerine 5 per cent, alcohol, 5 per cent, in 100 cc. distilled water and applied either as nose drops in the Proetz position or with an atomizer.

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* Supplied by Ciba Pharmaceutical Products, Inc., Summit, New Jersey.

† Supplied by American Ferment Co., Inc., 1450 Broadway, New York, 18, N. Y.

A mimeographed outline of the daily treatment, given to each patient, is shown as follows:

TREATMENT SCHEDULE.

DRUGS	*Morning*	*Afternoon*	*Evening*	*Bedtime****
Caroid Powder*				
Liquid				
Lamb's Wool (½ hr.)				
CAPSULES				
Antibiotics (Capsules)**				
(Aqueous)				
Lamb's Wool (½ hr.)				
TABLETS (after meals)				
Priscoline				
Niacin				
VITAMINS				

* Occasionally produces sneezing and blockage.
** Used with powder blower (De Vilbiss No. 119).
*** After 1 glass milk with crackers or toast.

Patients were instructed to omit treatment on their scheduled appointments so as to evaluate the clinical progress. Anosmia was appraised both from the subjective history and a blindfold test of various odors such as coffee, lemon, cooking spices, perfumes and alcohol. Metabolic, nutritional, and hematological problems were followed in consultation with the medical department of the Massachusetts General Hospital.

RESULTS.

In the combined total series of 35 patients, 17 (48.6%) showed striking improvement, 13 (37.1%) significant, three (8.6%) slight, and two (5.7%) no improvement. Patients classified as strikingly improved had a complete disappearance of odor, crusting and organisms of the klebsiella group. Local examination showed the return of normal sized turbinates with an increase in secretion, circulatory filling, and the sense of smell to a greater or lesser degree. In the group with significant improvement, there was a complete disappearance of odor, a marked decrease in crusts, some increase in the moisture and turbinal filling, and complete inhibition of the klebsiella organisms. There was no return of the sense of smell. Those considered slightly improved showed complete or partial disappearance of odor, slight reduction, in the crusts, and no change in the atrophy. The klebsiella organisms were inhibited to a variable degree.

More than 50 per cent of the strikingly improved patients were treated with streptomycin, but of these nearly one-half later required either the additional use of Chloramphenicol or Terramycin. In the remainder of the group, Terramycin was administered more often than Chloramphenicol. Those showing significant improvement needed more prolonged antibiotic therapy. The majority of these cases were benefited by Terramycin. In both the striking and significantly improved groups, aqueous antibiotics and Gantrisin were also used locally and orally depending upon the bacterial flora and clinical response obtained. Friedländer's bacillus was completely controlled, while B. coli was inhibited by 50 per cent. The two most resistant strains were H. staphylococcus aureus (37%) and B. proteus (14%), the latter showing less frequency after treatment.

In the follow-up of the 17 strikingly improved cases, seven have been able to maintain their continuous recovery without antibiotics or local treatment over a period of four months to approximately three years. Three of the seven patients with fully recovered turbinates have shown no recurrence of their atrophy after going without vasodilators from five months to nearly two years. The remaining 12 patients have continued in varying degree with vasodilator therapy and the hygienic use of powdered Caroid of 6 per cent and lamb's wool. No harmful side effects have been noted from the prolonged use of the antibiotics and vasodilators. Of the 12 patients in the strikingly improved group who showed marked atrophy (3+ to 4+) prior to vasodilator therapy, seven revealed turbinates that looked relatively normal in size and appearance after two and one-half months to one year of treatment. Six of the seven with less advanced atrophy (1+ to 2+) showed normal looking turbinates after one to six months of treatment. In 77 per cent of the 13 patients with normal looking turbinates, X-rays taken confirmed the clinical evidence of turbinal recovery (see Figs. 1, 1a). Although turbinal growth was not fully shown by X-ray in the remaining cases, it was clinically evident that increased turbinal filling preceded demonstrable X-ray changes.

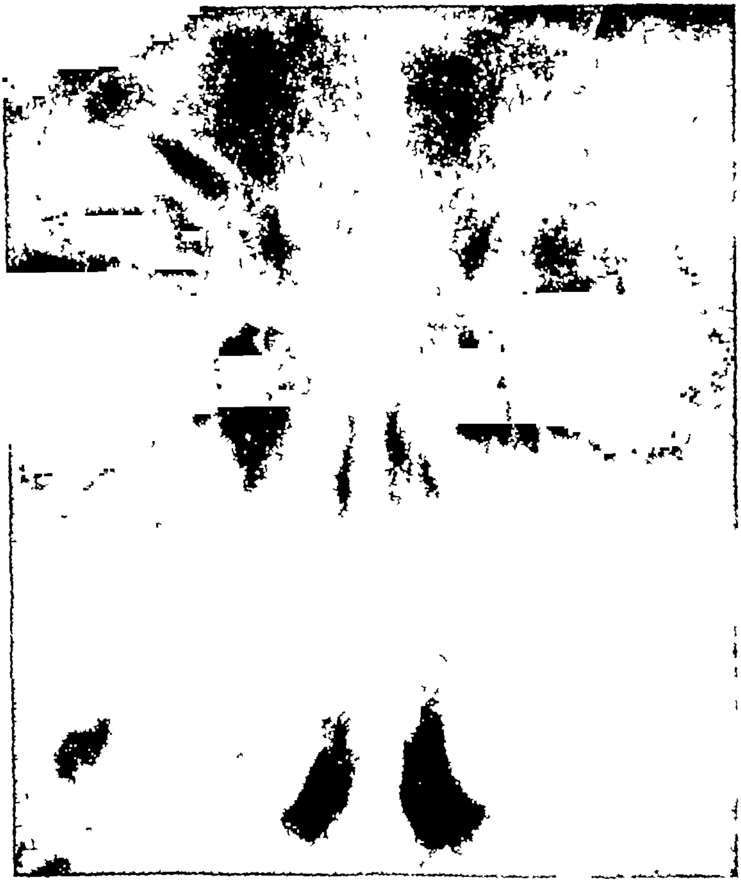

Fig 1 A. N Female Age 31 Before treatment. Marked Turbinal atrophy Sinuses negative. Case 13

Priscoline and niacin in combination appeared to be the vasodilator drugs of choice for promoting an increase in the circulatory filling and secretory functions of the atrophic turbinate. Only one out of five in the early group of 10 cases responded to the addition of histamine, while three out of the remaining five were strikingly improved without its use. In the newly added group of 25 patients, only three were given histamine combined with Priscoline and niacin. Two (5%) patients developed histamine sensitivity reactions with gastro-intestinal, skin and asthmatic symptoms after several weeks of treatment necessitating its elimination. Priscoline intolerance was seen to develop in eight (22.85%) of the 35 cases, although later attempts to resume the drug in some of these patients showed an increased tolerance. The determination as to how long vasodilator therapy should be maintained for its maximum effectiveness remains to be further evaluated.

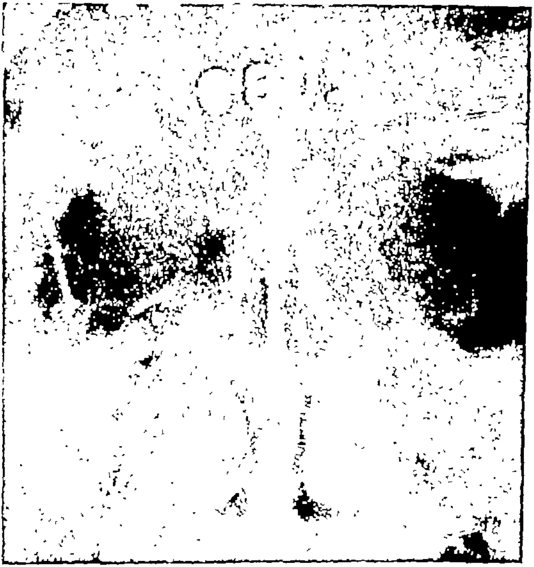

Fig. 1A. After 27 months treatment. Turbinates restored to normal appearance.

Of the nine patients with complete anosmia, four showed an appreciable return of their sense of smell and two merely a slight return.

Sensitivity tests revealed gram positive flora to be most responsive to Terramycin and bacitracin. B. Friedländer's, B. coli, and H. influenza were equally responsive to streptomycin, Terramycin and Chloramphenicol. B. proteus and B. pyocyaneus were preponderantly insensitive to the antibiotics. Sensitivity tests as a guide to local specific therapy, however, were generally found to be less accurate and reliable than the actual clinical response obtained.

FOLLOW-UP OF EARLIER CASES WITH AQUEOUS ANTIBIOTICS.

Ten of the 13 patients comprising the earlier series[15] of cases treated with aqueous antibiotics were followed for two to 13 months after the antibiotics were omitted. Vasodilator therapy along with the local use of Caroid and lamb's wool was continued. Two out of four patients in this group maintained their earlier striking improvement for three and 13 months respectively, while the remaining two showed some regression after two and one-half and six months. In the significantly improved cases, three out of four held their gain for two and one-half to three months. The remaining patient returned after an absence of two years with a complete recurrence of symptoms. Of the two with slight improvement, one showed a complete regression after five months. On the basis of these results, the use of insufflation therapy was investigated for a more effective local method of antibiotic administration and dosage.

FOLLOW-UP OF EARLIER CASES WITH ANTIBIOTIC INSUFFLATION.

Eight of the 10 patients were subsequently treated with streptomycin, Terramycin and Chloramphenicol for an additional period of two and one-fourth to 13 months and followed for six to 26 months after the antibiotics were omitted. Vasodilators were continued. Three recovered their earlier striking improvement and have held this result from nine months to two years after antibiotic insufflation was omitted. Two of these, were treated with Terramycin and one with Chloramphenicol. The three patients who maintained significant improvement, while making no further clinical progress showed little regression during the six months to more than one year after insufflation was terminated. One patient had been treated with Ter[illegible] with Terramycin and later streptomycin, and f[illegible] all three, of which Terramycin was the most [illegible] the two patients with slight improvement showed [illegible] despite prolonged antibiotic therapy from several m[illegible] approximately two years. One was treated with Terram[illegible] streptomycin combined, the other with Terramycin follo[illegible]er by Streptomycin. In the latter patient, vasodilator th[illegible] has been continued for as long as 33 months. This was [illegible] only patient whose cultures continued to show Friedländer's bacillus. No correlation was found between the culture changes in the bacterial flora and the clinical results except for the consistent inhibition of Friedländer bacillus in nearly all cases.

FOLLOW-UP OF LATER CASES WITH ANTIBIOTIC INSUFFLATION.

Insufflation therapy in [illegible] group of the 25 newly added patients was given at various intervals from one week to 15½ months. Combined treatment with vasodilators was simultaneously carried on over a period of 24 months. Thirteen of the 25 were strikingly improved. Ten showed significant, one slight, and one no improvement.

In about one-third of those showing striking improvement, antibiotics were given from one week to three months. In the remaining two-thirds, insufflation therapy was carried on from four and one-half to 10 months with the additional use, in some, of Gantrisin and aqueous antibiotics. All but five of the strikingly improved group of patients (61%) responded without further need of antibiotic therapy during the follow-up period of several months to more than one year. Priscoline and niacin were used throughout in combination except in one patient, where histamine was added.

In the significantly improved group, eight of the 10 patients were still under active treatment from two weeks to over one year. Priscoline and niacin were being given to some up to four months and others between four and nine months. In a few patients, these drugs were taken continuously for about one and one-half years, the longest in one case being approximately two years, with the additional use of histamine most of the time.

The follow-up cultures taken in this later series of cases showed no growth of either Friedländer's bacillus or B. coli; however, H. staphylococcus aureus was predominant, except in two patients where B. proteus and odor persisted.

DISCUSSION.

Incidence: There is no specific information available on the morbidity of the disease throughout the world. Recent estimates obtained from various colleagues, listed in the table below,[41] as to the incidence in their clinic and private practice during the past five years offer reasonable evidence of a high prevalence throughout Europe, particularly in the southern and eastern parts as well as the Near East. Gusic has observed an even greater incidence in Yugoslavia since World War II as compared with the number of cases seen up to 1950. Several of our resident house officers from the Near and Middle East state that the disease is especially prevalent among their desert and agrarian populations. Ersner[36] reports a similar incidence. Wodak of Israel, however, writes that the number of ozena cases among the natives is extremely small by comparison with the European immigrants in the last few years. The impression throughout England, France and the Americas, is that it has shown a steady decrease in these countries during the past 25 years. In the absence of known causes, various explanations given are at best speculative. Pollock (1934) believed it was due to a restriction of immigration from Southern and Southeastern Europe; also earlier treatment plus prevention of chronic sinus infections as exciting factors. Quevedo of Guatemala asserts the cause to be late hereditary syphilis and consequently on the decrease. Gusic, however, observes that ozena is practically non-existent in the Dinar Highlands of Yugoslavia where lues is known to be endemic. In Cuba, Basterrechia believes the diminishing incidence is due to the yearly increase of diphtheria immunization in children. In Peru, Raffo attributes it to the improvement in hygienic and living conditions. The statistical estimates thus far received[†] and their geographic distribution are shown as follows:

ESTIMATES ON INCIDENCE FOR THE PAST 5 YEARS (1947 to 1953)

	Region	Hospital	Private Practice	Comments
United States				
Blaisdell, I. H.	N. Y.		12	
Daly, J. F.	N. Y.	15*	8**	* Bellevue Hospital ** 1953
Ruskin, S. L.	N. Y.		15–25	
Ersner, M. S.	Pa.	22*		* Temple Univ. and Jewish hospitals. Prevalent in Slovak group.
Henner, R.	Ill.		30–40	
Lederer, F. L.	Ill.		1* 5* 20*	* From associates. One per 500 Chinese recruits. (L)
Proetz, A. W.	Mo.		60	More prevalent in indigent foreign born population.
Brown, L. A.	Ga.		11	
Simonton, K. M.	Minn.	5		1949, Mayo Clinic
Thornell, W. C.	Ohio	10*	3	* 1946, Mayo Clinic
Lierle, D. M.	Iowa	35		University Hosp.
Brown, J. M.	Calif.		5	
Fitzhugh, W. M.*	Calif.			* Occurs in 5% of practice.
Morrison, L. F.	Calif.		42	
West Indies				
Gros, J. C.	Cuba, Havana	29* 14* 6***	2	* 1925, Univ. of Havana Hospital. ** 1945 *** 1952
MacCormick, C.	Puerto Rico,	10	5	
Font, J. H.	Santurce		24	
Pico, J.			15	
C. and S. America				
Quevedo, J.	Guatemala	276* 213*	127* 115	* 1943-47 Assumed cause late hereditary syphilis.
Raffo, E.	Peru, Lima	15*	5	* Hospital Obrero. Decrease due to better living conditions.

[†] A more complete list will be reported later pending further replies from other countries.

ESTIMATES ON INCIDENCE FOR THE PAST 5 YEARS (1947 to 1953)
(Continued)

	Region	Hospital	Private Practice	Comments
C. and S. America (Cont.)				
Romero-Diaz, E.	Argentina, Cordoba	8–9		Hospital Cordoba.
Tato, J.	Buenos Aires	10		
Gutierrez, C.		7		
Jahn, F. C.	Venezuela* Caracas			* Exceedingly rare—due to antibiotics. Assumed etiology—B. pseudo-diphtheria.
Kos, J.	Brazil, Rio de Janeiro		60	Decrease noted.
Tello, A.	Chile, Santiago	130*		* Men, 28; women, 102.
Europe; Near East				
Negus, V. E.	England, London		10–15	
Ormerod, F. C.	London	107*	26**	* 1952, Inst. of Laryngol.-Otology. ** 1944-48
Portmann, G.	France, Bordeaux	60		* Clinique d'Oto-Rhino-Laryngologie.
Ruedi, L.	Switzerland, Zurich	187		Kantonsspital
Meurman, Y.	Finland, Helsinki	116		OPD not included.
Zollner, F.	Germany, Frieburg	82		Universitats-Klinik
Demetriades, Th.	Greece, Athens	451*		* Clinic cases, 1943-53
Gusic, B.	Yugoslavia, Zagreb	134*		* 1950-53
Wodak, E.	Israel, Tel-Aviv			Extremely rare among natives as compared with European immigrants since 1939
Australia				
Pettinger, D.	Sydney, Camperdown	6*		* Royal Alexandra Hosp. OPD.
Africa	Johannesburg	60–100		Non-European Hosp. OPD.

Although these figures represent random samples, they are, nevertheless, of sufficient significance to indicate the scope and challenge of the problem.

Odor: Despite various hygienic precautions of snuffing salt-water, daily nasal douches and oily sprays previously taken for many years, all patients were acutely apprehensive of their odor, whether actually present or not. The fear of being rejected as social outcasts was sufficiently provocative to channelize many deep-seated anxieties into a variety of psychosomatic symptoms. Recognition of these emotional problems and constant reassurance were important considerations throughout the entire course of treatment.

Hygienic Management: Allergic-like reactions of nasal obstruction, sneezing, ear blockage and asthmatic chest symptoms were occasionally encountered with the use of powdered or liquid C[illegible]id (6%). Complete intolerance to the drug was present in [illegible] per cent of the cases.

Local Antibiotic Therapy: In comparison with aqueous therapy, antibiotic insufflation resulted in longer follow-up periods without regression after their discontinuance. Allergic skin or mucous membrane reactions were less encountered than with the aqueous antibiotics. Streptomycin in high dosage ([illegible]00 mgs. once daily) appeared to promote in some a vascular hyperemia and slight bleeding of the nasal mucosa which was readily controlled with smaller or divided doses, *i.e.*, 50 to 100 mgs. twice daily.

The prolonged use of Gantrisin in both local and oral form was considered in general, effective against the resistant B. proteus organisms. Bacitracin proved efficient in its action upon gram positive flora without side effects wherever aqueous therapy seemed indicated. In the earlier group of cases, penicillin sensitivity of the nasal and oral mucous membrane was observed in over one-third (38%) of the cases from its aqueous use within a few weeks of local treatment. Only two (15%) showed allergic sensitivity to aqueous streptomycin, one of these after the fifth month in combination with penicillin.

Vasodilator Therapy: The immediate vasomotor response varied in its duration from several minutes in some to one hour or longer in others. It consisted of either a hot flush or a tingling sensation of the scalp. Most patients noted a gradual increase in their body warmth, particularly in the hands and feet. In one case not included in this series, an irregularity of the menses was observed with Priscoline, returning to normal after the dosage was reduced. The use of the vasodilator drugs proved more effective in combination than singly for promoting the circulatory response and secretory function of the nasal mucosa. An indication of tissue permeability was evident from the resulting local manifestations such as, an increase in the erectile-tissue filling of the inferior turbinates, greater moisture content of the nose, the partial return of the sense of smell, and the local effectiveness of antibiotic therapy. It was not anticipated that turbinal recovery would take place where an atrophy of many years' duration had finally resulted in marked skeletonization. Nevertheless, even in this advanced stage, atrophic cellular changes were not entirely irreversible, since their histological improvement was observed after prolonged therapy.

Surgical Measures: In two cases with marked deviations of the septum, replacement surgery was additionally performed with submucous cartilage implants filling in the concavity defect to increase resistance to nasal airflow and, thereby, condensation within the nasopharynx. Resorption of the implant resected from the upper part of the patient's septum was observed in one case.

Results: The highest percentage of patients with striking improvement occurred in approximately 75 per cent to 60 per cent between the second and fourth decades. The percentage dropped from 50 per cent to 20 per cent between the fifth and seventh decades. The figures are illustrated as follows:

Years	Total Number Patients	RESULTS			No
		Striking	Significant	Slight	
0–10	1	0	1	0	0
10–20	4	3	0	0	1
20–30	5	3	2	0	0
30–40	8	5	2	1	0
40–50	4	2	2	0	0
50–60	10	4	4	1	1
60–70	2	1	1	0	0
70–80	1	0	0	1	0

In the strikingly improved cases, the earliest elimination of odor, crusts and discharge became evident from three to eight weeks with antibiotic insufflation. A decrease in turbinal atrophy was usually apparent after the third month with vasodilator therapy. As might be expected, the response was much slower in patients with significant improvement and in those who had had previous sinus surgery.

Thus far, the evidence based on the results reported in this paper offers no particular etiology but rather suggests that ozena may be a target disease which viewed in terms of successful therapy postulates a sustained overall approach from various directions both local and systemic. Most of the cases indicated a combination of symptoms pointing to metabolic, nutritional, and hematological deficiencies.

SUMMARY.

A new method for the treatment of ozena is presented by combining the local insufflation of various micronized antibiotics, such as Streptomycin, Terramycin, or Chloramphenicol (Chloromycetin) with the multiple use of certain oral vasodilators, Priscoline and niacin. Dosage and administration are described, including the additional use of aqueous bacitracin. Streptomycin, or Gantrisin. Sodium lauryl sulfate, a surfactant, and beta lactose, an acid vehicle, are incorporated with the micronized antibiotics for their greater effectiveness.

The management of nasal hygiene with the combined use of lamb's wool plugs and liquid or powdered Caroid 5 to 6 per cent (papaine) is carried out as a supportive measure to increase nasal moisture and dissolve encrusted secretions, thus eliminating the need for nasal irrigations.

A total series of 35 selected cases observed over a period of three years are reported. The results show 17 (48.6%) with striking improvement, 13 (37.1%) significant, three (8.6%) slight, and two (5.7%) with no improvement.

Three case reports with X-ray and microscopic evidence of turbinal growth and regeneration of respiratory epithelium are presented. The literature is reviewed.

CONCLUSIONS.

The prolonged use of micronized antibiotics for insufflation therapy in ozena causes no deleterious effects and constitutes a safe procedure.

Vasodilator drugs prove more effective in combination than singly for promoting the circulatory response and secretory function of the nasal mucosa. Turbinal recovery precedes demonstrable X-ray changes.

The combined use of properly selected antibiotics and vasodilators offers a promising method of treatment for the effective elimination of odor and crusts and a reversal of the regressive changes in ozena.

ILLUSTRATIVE CASE REPORTS.

Case 2, Later Series: B. N. A 26-year-old male, was first seen in the E N T Clinic (1945) with symptoms of "sinus trouble" since boyhood.

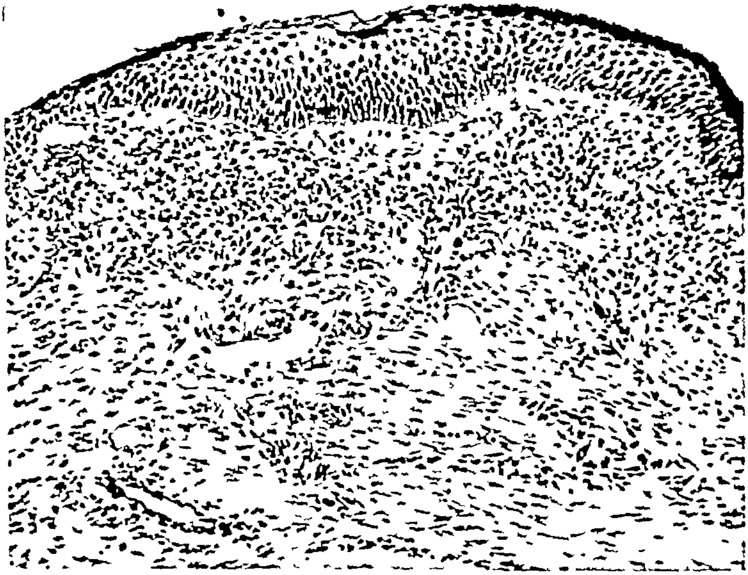

Fig 2 B. N Male Age 26 Before treatment. Inferior turbinate (mid-third section) showing stratified squamous epithelium Basement membrane and tunica propria appear within normal limits Ill-defined subepithelial vascular spaces with no fibrosis Infiltration moderate. High power (130 X). Case 2. (Later series).

The nasal findings were recorded as odor, discharge and turbinal atrophy. There was a previous history of a submucous resection in 1938. X-ray of the sinuses showed pus in the right antrum and chronic thickened membrane in both ethmoids and antra. A right antrum wash disclosed a slight amount of pus. Nasal irrigations and an oily spray (iodine, menthol, camphor) were prescribed.

He returned for treatment six years later in April, 1951, and was referred to the atrophic rhinitis clinic for study. Examination revealed marked blockage, odor, green crusts, discharge, an anterior septal perforation (post-operative), moderate turbinal atrophy and anosmia. The culture reported was B. proteus. X-ray of the sinuses now showed no disease except for intranasal atrophy. A biopsy (see Fig. 2) revealed stratified squamous epithelium with a normal basement membrane. The lymphocytes and plasmocytes appeared within moderate limits and fibrosis of the tunica was not unduly dense. No glands were evident. The vascular spaces subepithelially appeared ill-defined showing neither fibrosis nor infiltration except for deeper thin-walled vessels.

Insufflation Therapy: Powdered Caroid (6%) and lamb's wool were prescribed for one month without appreciable effect. Insufflation with Streptomycin and later Chloromycetin, alternating with Gantrisin (oral and local), was carried on for over seven months.

Result: Striking clinical and histological improvement were noted with an increasing return of the sense of smell. A

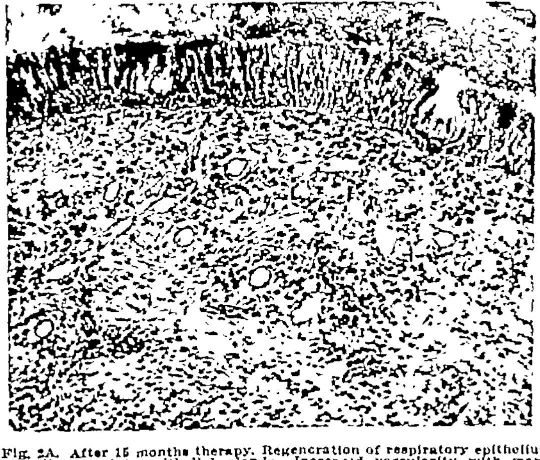

Fig. 2A. After 15 months therapy. Regeneration of respiratory epithelium with cilia and intraepithelial glands. Increased vascularity with many well defined capillaries superficially and larger vessels of normal appearance. High power. (130 X).

follow-up period for 10 months without antibiotics showed no regression. Priscoline and niacin, with the less frequent use of Caroid and lamb's wool was continued for a total of 15 months. Pneumococcus and H. staphylococcus aureus were reported in the last culture. X-rays taken after 14 months of treatment, June, 1952, revealed no demonstrable change in the atrophy. A biopsy (see Fig. 2A) after 15 months of therapy, July, 1952, showed regeneration of respiratory epithelium (Pseudo-stratified columnar ciliated epithelium) with intra-epithelial glands. The basement membrane and tunica propria appeared within normal limits as before except for a few newly observed glands. Many vascular spaces with well marked walls were seen as compared with their thin walled structure previously described. There were numerous capillaries and larger vessels of normal appearance.

Case 2, Earlier Series: P. C. A 53-year-old woman with a history of ozena dating back to childhood was first seen in the E.N.T. Clinic in 1937. Examination revealed odor, crusting, atrophy and anosmia. X-rays

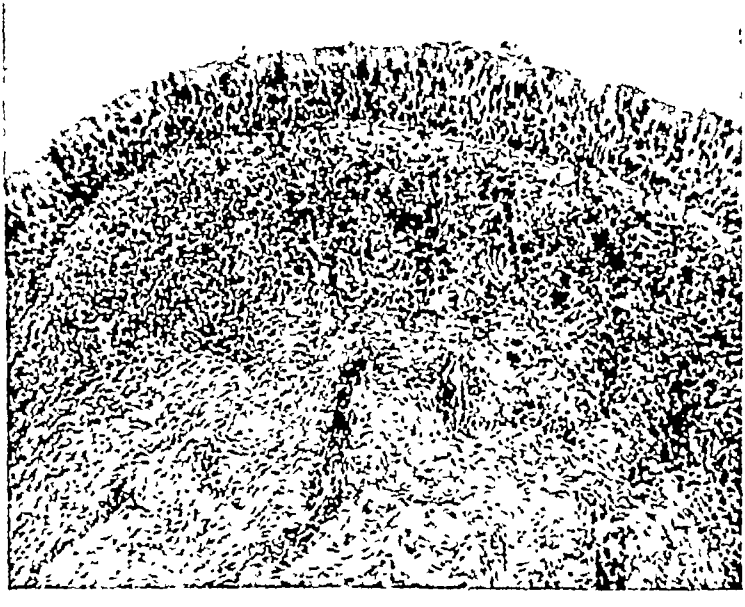

Fig. 3. P. C. Female Age 53. After 12 months therapy. Inferior turbinate (mid-third section) showing respiratory epithelium, marked infiltration and fibrosis No goblet cells present. A few capillaries are evident but not well marked. Small blood vessels appear constricted by fibrous tissue. High power. (130 X). Case 2 (Earlier series).

of sinuses were negative. In 1938, a bilaterial Caldwell-Luc was performed for a bilateral chronic suppurative pansinusitis followed by estrogenic nasal sprays until 1946 with no improvement. X-rays still showed a bilateral chronic suppurative pansinusitis with marked intranasal atrophy. A revision bilateral Caldwell-Luc and intransal ethmoidectomy was again followed by a persistence of symptoms.

Combined treatment with aqueous streptomycin, histamine and niacin was started in February, 1950. Bacitracin was employed later due to penicillin sensitivity. Results after eight and one-half months of continuous therapy showed striking clinical improvement with complete inhibition of Friedländer's bacillus. Antibiotics were omitted for three months and vasodilators, lamb's wool and powdered Caroid (6%) continued. Tenacious secretions soon recurred in the nasopharynx, however, with no odor or crusting. H. staphylococcus aureus was reported in the culture. A biopsy (see Fig. 3) taken 12 months after therapy, showed respiratory epithelium. No goblet cells were present. The basement membrane appeared slightly thickened, and the tunica revealed marked evidence of chronic inflammation with lymphocytic and plasmocytic infiltration and fibrosis. Only one gland tubule was seen. There were many vascular spaces deep in the tunica propria with a normal number of small blood vessels constricted by fibrous tissues and a few capillaries not well marked.

Comment: Unfortunately a "before" biopsy was not obtained. (This also applies to the following case report, Case

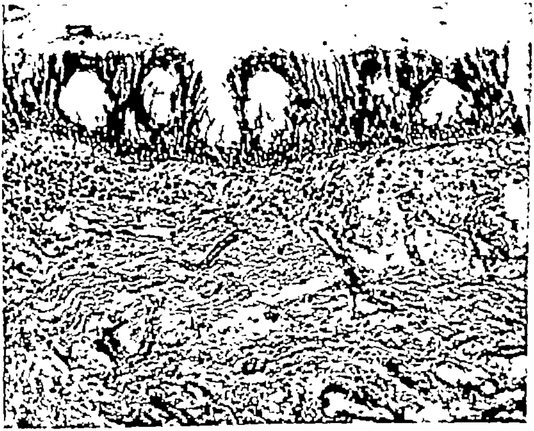

Fig. 3A. After 20 months' therapy. Definite regeneration of typical respiratory epithelium with goblet cells. Marked reduction of previous infiltration with increased vascularity of vessels close to the surface. Large spaces in epithelium are probably intraepithelial glands. Note well defined cilia. High power. (130 X).

13). The second biopsy, however, taken considerably later, showed further evidence of regenerative change.

Insufflation Therapy: Terramycin was prescribed for insufflation during the next three months.

Results: Striking clinical and histological improvement was observed with a return of the sense of smell. A follow-up period for 18 months without antibiotics showed no regression. Niacin, lamb's wool, and the occasional use of powdered Caroid were continued. H. staphylococcus aureus was last reported. X-rays taken in July, 1952, after two and one-half years of treatment revealed turbinal enlargement with a general reduction in atrophy. A second biopsy (see Fig. 3A) now showed definite respiratory epithelium with goblet cells and relatively little evidence of active infection in the tunica propria. One gland tubule was seen, also several new capillaries. The larger blood vessels appeared normal.

Case 13, Earlier Series: A. N. A 31-year-old woman was referred to the atrophic rhinitis clinic in April, 1950, with a history of ozena symptoms of 10 or more years' duration. Examination revealed considerable odor, crusting and anosmia with moderate atrophy. X-ray of the sinuses (see Fig. 1) showed marked atrophy of the turbinates but otherwise were negative. A culture was reported beta H. streptococcus, H. staphylococcus aureus and B. proteus.

Combined treatment was started with aqueous Streptomycin, Penicillin, Priscoline and niacin. Penicillin was omitted after one month because of sensitivity and aqueous Gantrisin 20 per cent was used more or less continuously for seven months without successful inhibition of B. proteus. After nine months therapy, there was significant improvement with clean nasal airways and normal appearing turbinates. An objectionable odor, however, was detectable due to persistent crusting in the nasopharynx. A biopsy at this time, showed both columnar epithelium .and stratified squamous epithelium, corresponding to the lateral and medial surfaces respectively in the same specimen. The basement membrane in both appeared slightly thickened. There was slight to moderate cellular infiltration and fibrosis. The glands were slightly atrophied, and many small vessels showed perivascular fibrosis. Capillaries were scanty.

Insufflation Therapy: Chloromycetin insufflation was employed intermittently for nine months during the following year.

Results: Striking clinical and histological progress with a complete return of the sense of smell was observed over a follow-up period for eight months without antibiotics. The culture reported was a resistant H. stapyhlococcus aureus and B. proteus. Treatment was maintained with niacin, lamb's wool and only the occasional [illegible] [illegible]ered Caroid due to the blockage reaction produ[illegible] [illegible] was employed intermittently because of sid[illegible] [illegible]ays (see Fig. 1A) taken in July, 1952, follo[illegible] [illegible] treatment showed turbinates of normal [illegible] [illegible]nd biopsy revealed a regeneration of res[illegible] [illegible] Goblet cells were not clearly identified. The [illegible] [illegible]mbrane and tunica propria appeared nor[illegible]. There were [illegible]ny small capillaries and larger vessels of [illegible]mal appearance.

The author is gratefu[illegible] to Dr LeRo[illegible] A. Schall, chief of staff, and to the following mem[illegible] their kind assis[illegible]ce and cooperation: Dr. Werner Mueller, Dr. A[illegible] S. Macmillen, [illegible]s Anita Mangiaracine, bacteriologist, Mr. Jo[illegible]phy, pharmacolog[illegible] Mr. Charles Snyder, librarian, and Miss [illegible]etrone, secretary. [illegible]e author also acknowledges with apprecia[illegible] generous assistan[illegible]nd special studies conducted by Dr. Lesli[illegible]hmart of the Massac[illegible]usetts College of Pharmacy, in the form[illegible] of the antibiotic [illegible]parations used in this project.

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MEETING OF THE GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The Georgia Society of Ophthalmology and Otolaryngology will meet March 11-12, 1955, at the General Oglethorpe Hotel, Savannah, Georgia.

The speakers will be: Dr. Francis H. Adler, Philadelphia; J. W. McCall, Cleveland; Dr. J. A. Hilger, St. Paul; Dr. Walter H. Fink, Minneapolis; Dr. James H. Allen, New Orleans; and Dr. P. E. Ireland, Toronto, Canada. For further details, address Alton V. Hallum, M.D., Secretary, 245 Doctors Bldg., Atlanta 3, Ga.

HYDROCORTISONE ACETATE AND NEOMYCIN IN OTIC INFECTIONS.*†

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I. Introduction.—

In this era of new and remarkable chemotherapeutic agents and antibiotics, it is not unusual to find reports of excellent results in the treatment of recalcitrant otic infections; however, there have been few topical medications introduced in recent years which have proved of lasting value in the treatment of chronic and recurrent irritations in and around the external ear. Neomycin and hydrocortisone acetate (compound F) in proper vehicles seem to offer a solution to some of these difficult otologic problems.

Some of the most resistant infections and dermatoses found in the region of the external ear include simple pruritus or pruritus auris, localized neurodermatitis, postoperative infections of the skin lining the mastoid and fenestration cavities, and otitis media. These cases offer many problems as the physician attempts to control the primary infection as well as the secondary diffuse external otitis or infectious eczematoid dermatitis produced by the persistent drainage.

These dermatoses have been treated with some success with a variety of topical antibiotics and chemotherapeutic agents; however, these lesions are very prone to develop chemical or allergic reactions. In the mastoid and fenestration cavities, in particular, there has been a high incidence of fungus disease superimposed upon the original lesion as a result of the

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insufflation of various antibiotics; thus, some preparations have been helpful for a short period, but very few have demonstrated consistent success.

Radiation therapy is one of the techniques often used to control neurogenic lesions in and around the ears. This has offered excellent temporary improvement, but its frequent use is fraught with danger and, therefore, is used reluctantly by many otologists and dermatologists.

II. Literature Review.—

There have been many reports describing excellent results from the use of hydrocortisone acetate for various dermatoses.[3] In histopathologic studies of sundry skin inflammations it was demonstrated that *compound F inhibited the inflammation of eczematous, toxic, and some miscellaneous skin disorders.*[4]

Neomycin has been shown to have a marked bacteriostatic and bactericidal effect upon a wide range of bacteria,[7] including [illegible] variety of Gram-positive and Gram-negative *organisms* and actinomycetes. *It appears to be active against certain* strains [illegible] E. coli[8] and Proteus[9,10] *and is effective against some* streptomycin-resistant *strains of bacteria.* Bacterial *resistance* develops *more slowly than to other antibiotics,* and no absolute resistance was demonstrated. On local application there is limited toxicity, but when given systemically it has been shown to be nephrotoxic. Neomycin is not effective against fungi or viruses.

Because of its broad coverage, neomycin in various water miscible and greasy bases has been used widely over the past few years for the topical treatment of a variety of dermatitis and dermatoses. It has been shown in comprehensive studies[11,12] that the therapeutic results with neomycin are superior to those with previous preparations. Patients with atopic dermatitis, stasis dermatitis, and seborrheic dermatitis were very tolerant to neomycin, but those with hemolytic streptococcus and pyocyaneus infections failed to respond satisfactorily.[11,13] In another study[12] a variety of conditions including impetigo, folliculitis, and impetiginized eczematous dermatitis were treated with impressive results.

Of particular importance is the low index of sensitivity described by various investigators. This preparation can be used in the highly sensitized areas without too much concern regarding secondary reactions even in those instances where reactions have occurred previously. One investigator reported localized cutaneous moniliasis following the application of neomycin;[13] another described a contact type sensitization apparently occurring five months or more after the beginning of treatment.[14]

Recently, Sulzberger and Rein[15] reported that hydrocortisone ointment topically applied to the ear was effective in relieving inflammation and itching in some cases of external otitis. Baer and Litt[16] suggested the use of a hydrocortisone-neomycin suspension and described the results in ten cases of external otitis of various and diverse morphologic and etiologic types.

III. Methods and Procedures.—

An accurate differential diagnosis of the type of external otitis present is essential, since it will be shown that this combination of drugs is very effective for certain types of inflammations of the external ear, while of questionable value in the treatment of other forms. Diseases of the external ear have been classified on an etiologic basis,[17] and cases are so divided in this study. Following are brief definitions of the various categories of diseases treated. Some overlap of categories is always present and will be commented upon later.

1. *Neurogenic External Otitis.* The largest number of cases in this category are classified as simple pruritus. This is predominantly an affliction of middle-aged women. Such persons are tense, hyperactive, and are very voluble about their sufferings. There is usually intense itching without visible lesions. They tug and scratch their ears and use any available instrument to relieve the persistent itching sensation. Cultures show a predominance of Gram-positive organisms although Gram-negative bacilli are seen occasionally.

   A small number of cases of localized neurodermatitis are included. These are characterized by various sized circumscribed, elevated, excoriated, scaly patches in which there is thickening and an increase of skin markings. Other isolated plaques may be seen occasionally on the sides and back of the neck, eyelids, and anticubital areas.

Diffuse External Otitis. This is an acute or chronic symptom complex present mainly during hot, humid weather. The mild acute case has slight pain on manipulation of the auricle. The skin of the ear canal reveals some edema, slight redness and a coating of odorless, adherent secretion or exfoliated debris. In the more severe acute cases, patients complain of intense pain on mastication and on manipulation of the external ear. There is marked periauricular edema and partial or complete obliteration of the canal lumen by the edematous walls. Gray or green sero-purulent secretions and sheets of exfoliated debris are seen in the remaining lumen. The skin of the canal is thickened, purplish red in color and may have a papular appearance resembling gooseflesh, particularly on the superior and inferior walls.

In the chronic case a diffuse thickening of the skin of the entire canal is found. There may be a fetid gray-brown or greenish secretion coating the skin and filling the tympanic recess. Papules and vesicles are sometimes seen, and the drum loses its luster, becomes thickened and shows some loss of detail.

Cultures of the acute and chronic cases almost invariably show an abundant growth of Gram-negative bacilli although occasionally fungi are found. Stained smears reveal myriads of bacilli and epithelial cells.

Postoperative Dermatitis of Mastoid and Fenestration Cavities. In a small number of cases drainage persists from the postoperative mastoid or fenestration cavities despite active therapeutic measures. Cultures usually show a predominance of Gram-positive organisms.

4. *Otitis Media.* In this group are included subacute and chronic infections of the middle ear and possibly the mastoid antrum from which a mixed bacterial flora of Gram-positive and Gram-negative organisms is usually obtained.

5. *Otomycosis.* Diseases falling under this heading are chronic or recurring. The patient complains of intense itching, a feeling of fullness in the ear, difficulty in hearing and later, severe pain. Examination may reveal moist cerumen intermixed with exfoliated scales and sheets of epithelium, or the depths of the canal may be filled with wet grayish "blotting paper" debris. In uncomplicated cases the organisms may be seen as green, black, or white filamentous structures. Direct wet unstained smears reveal the presence of mycelia, spores, neutrophiles, and myriads of epithelial cells.

Hydrocortisone acetate ointment (1.5%)[*] was applied to all neurogenic lesions present in the concha, at the meatus or in and around the auricle. The patient was instructed to remove the adherent crusts or scales with mineral oil or vaseline and then to apply the ointment three times a day. If weeping and vesiculation were present, mild aluminum acetate packs were used as a cleansing astringent prior to the application of the ointment.

Neomycin-hydrocortisone acetate drops (1.5%) in an aqueous vehicle[*] were used in all post-fenestration and post-mastoid cavities and in patients with otitis media. Prior to the institution of therapy, the cavity was cleansed with hypertonic saline irrigations, and all exfoliated debris was removed by forceps or suction. Granulations were curetted away, and any evidence of fungi was treated with proper fungicidal agents. The patient was instructed to lie on the unaffected side, instill five drops of the suspension into the external ear for five minutes, three times a day. In occasional cases the ointment was applied to a wad of cotton which was then packed in the mastoid cavity and allowed to remain for 24 to 48 hours.

Irrigations were repeated on follow-up visits as often as necessary in order to keep the cavity clean and permit the therapeutic agent to come in close contact with the irritated or infected skin. Cultures and smears were obtained both pre- and post-treatment as often as possible, and the bacteriologic changes recorded.

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[*] Neocortef ointment containing 15 mg. of hydrocortisone acetate compound F and 5 mg. of neomycin sulfate per gram, and Suspension of Neocortef acetate (1.5%) containing 15 mg. of hydrocortisone acetate compound F and 5 mg. of neomycin sulfate per cc. were generously supplied by the Upjohn Co., Kalamazoo, Mich.

IV. Results and Discussion.—

The pertinent data and findings of the 90 treated patients (114 ears) are summarized in Tables 1 and 2. It is noteworthy that the average age of all groups is older than one would anticipate. There seems to be a predominance of females in the neurogenic and acute diffuse groups, but no predilection for one or the other ear.

The most significant clinical observation is the consistent and excellent response obtained in that group of indolent and recalcitrant ears placed in the neurogenic category. Only in occasional cases were indifferent results obtained.

The response of the patients was enthusiastic in that there was prompt control of the itching, vesiculation and weeping. Mild exacerbations were treated easily by the patient; thus the necessity for radiotherapy was eliminated in every case in this series. Needless to say, exacerbations occurred in those cases where the psychosomatic factors contributed to the production and persistence of the lesion. In these cases efforts were made to acquaint the patient with the underlying psychogenic basis for the dermatosis.

Of special importance to the otologist is the remarkable response obtained in the treatment of the persistently draining post-operative mastoid and fenestration cavities. Cavities which had been draining for years and showed ulceration and granulation suddenly responded to the application of this form of therapy. The skin lining which had appeared reddened, thickened and unhealthy, rapidly assumed a healthy color. Cavities which had shown granular, weeping islands, particularly on the tegmen mastoidea, responded promptly to treatment.

A number of unexplained failures with this form of therapy occurred in this latter group until previously unidentified fungi were found on smear or culture. Under these circum-

TABLE 1.

SHOWING DATA PERTINENT TO THE TREATMENT OF VARIOUS OTIC DISEASES WITH PREPARATIONS OF NEOMYCIN AND HYDROCORTISONE ACETATE.

Category	Average Age	No. Pts.	No. Ears	Ear Involved Rt.	Ear Involved Left	Sex F	Sex M	Form of Medication O	Form of Medication D	Form of Medication O & D
N.E.O.	40 yr.	22	32	16	16	22	10	21	4	7
A.D.E.O.	35 yr.	22	28	15	13	17	11	7	10	11
C.D.E.O.	48 yr.	6	10	5	5	5	5	1	4	5
O.M.	40 yr.	16	18	11	7	8	10	0	17	1
P.O.M.	24 yr.	9	9	2	7	6	3	0	8	1
P.O.F.	44 yr.	4	4	2	2	3	1	0	3	1
Otomycosis	26 yr.	8	9	5	4	5	4	2	7	0
Miscellaneous	20 yr.	3	4	1	3	1	3	1	0	3
Totals	35 yr.	90	114	57	57	67	47	32	53	29

N.E.O. —Neurogenic External Otitis.
A.D.E.O. —Acute Diffuse External Otitis.
C.D.E.O. —Chronic Diffuse External Otitis.
O.M. —Otitis Media.
P.O.M. —Postoperative Infection of Mastoid Cavity.
P.O.F. —Postoperative Infection of Fenestration Cavity.
Misc. —Perichondritis, Infantile Dermatitis, etc.

O—Ointment of Neomycin-Hydrocortisone Acetate.
D—Drops of Suspension of Neomycin-Hydrocortisone Acetate.

TABLE 2.

SHOWING DATA PERTINENT TO THE TREATMENT OF VARIOUS OTIC DISEASES WITH PREPARATIONS OF NEOMYCIN AND HYDROCORTISONE ACETATE.

Category*	No. Pts.	No. Ears	Predominant Bacteria Gram-Positive	Gram-Negative	No Growth	No Culture	Exc.	Response Good	Fair	Poor
N.E.O.	23	32	10	9	3	10	14	16	1	1
A.D.E.O.	22	28	4	16	1	7	11	12	2	3
C.D.E.O.	6	10	1	9	0	0	3	5	0	2
O.M.	16	18	9	6	1	2	10	5	0	3
P.O.M.	9	9	6	1	2	0	3	4	1	1
P.O.F.	4	4	2	1	0	1	2	0	1	1
Otomycosis	8	9	2	2	1	4	0	3	1	5
Miscellaneous	3	4	3	1	0	0	0	3	0	1
Totals	90	114	37	45	8	24	43	48	6	17

* See Table 1 for key.

stances all medication was stopped, and fungicides were applied for several days. Neomycin and hydrocortisone drops alone or a combination of drops and ointment was then used with rapid improvement.

The response to treatment of chronic otitis media cases was gratifying. The granulations appeared to shrink, revealing the periphery of the perforation and a view of the middle ear. In some of the cases it was then possible to nibble away a good part of the granulations. This allowed further penetration of the drops and further drying and shrinking of the middle ear tissues. In several cases large masses of cholesteatomatous debris could be removed with a small otic suction leaving dry middle ears.* It is to be noted that three failures occurred, and completely dry ears were not obtained in five others.

It was not anticipated that neomycin and hydrocortisone therapy would be highly successful in diffuse external otitis; therefore, in the early part of the study this type of treatment was used only when there was a secondary neurogenic element superimposed on the diffuse external otitis. The response was sufficiently good so that it was used for uncomplicated diffuse external otitis with relatively good results.

It is suggested that this is the treatment of choice wherever a question of differential diagnosis arises or where there are overlapping types. It is our belief that most antibiotics are contraindicated in the presence of a weeping erythematous lesion at the meatus. Whether primary or secondary to otitis media or diffuse external otitis, there is a high incidence of chemical or allergic reactions to "otic preparations" in these cases. In such instances, the safer combination of neomycin and hydrocortisone preparation appears indicated.

There are still many forms of external otitis which do not fall easily into clear etiologic or morphologic categories. Such cases have been placed in a small miscellaneous group. One of these, a severe perichondritis with superimposed pyocyaneus infection, responded nicely to packing with the ointment preparation.

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* Such ears will be observed carefully for possible recurrence because of the probability of residual cholesteatoma.

It is worthy of note that Gram-negative organisms, primarily E. coli and Pseudomonas, were cultured in more than half of the neurogenic ears. No streptococci were present among the Gram-positive organisms. It was not unexpected that almost all the cultures from the diffuse external otitis group showed predominant growth of Gram-negative bacilli.

The findings in this study confirm previous reports that cultures from the persistently draining postoperative mastoid and fenestration cavities usually show Gram-positive organisms.[18] Fungi are cultured in a surprisingly high percentage of these cases;[19] in fact, most of the patients listed under otomycosis are postoperative cases which responded poorly until culture reports revealed the presence of fungi.

The presence of a high percentage of Gram-negative bacilli in the cultures obtained from cases of chronic otitis media and mastoiditis is consistent with previous observations.[10] The excellent response of some chronic ears from which Proteus was cultured was not entirely unexpected in view of the reported *in vitro* effectiveness of neomycin.

In general this study points up the increasing necessity for the otologist and dermatologist to note clearly the differential diagnosis of diseases of the external ear. Only by appreciating the morphologic differences and the bacterial flora usually present in these various categories of diseases of the external ear can prompt and proper therapy be applied.

V. Summary.—

1. Ninety patients (114 ears) with various otic infections were treated with an aqueous suspension and/or an ointment containing neomycin and hydrocortisone acetate (compound F).
2. Consistent and excellent clinical results were obtained in the treatment of the neurogenic type of external otitis.
3. Recalcitrant infections of post-fenestration and post-mastoidectomy cavities showed remarkable response to the aqueous form of therapy.
4. The response of a high percentage of patients with chronic otitis media, acute and chronic diffuse external otitis was gratifying.

5. Otomycotic lesions failed to improve until the fungi were identified and eradicated.

We gratefully acknowledge the referral of post fenestration patients by Dr. Theo. E. Walsh and the technical assistance of Nancy Hellman.

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THE EVALUATION OF HEARING IN PRE-SCHOOL CHILDREN, WHO LACK NORMAL SPEECH. A PRELIMINARY REPORT.

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The child who has not learned to speak by the age of three years is commonly presumed to be retarded, or hard of hearing. There are, however, four major groups of children who have, in common, lack of normal speech at that age, namely, (1) The Deaf or Hard of Hearing; (2) The Brain Injured, including those with Aphasia; (3) The Autistic, or those with a severe psychic disturbance, and (4) The Mentally Retarded. It is obvious that an early diagnosis is essential for the proper medical, psychological and educational rehabilitation of these exceptional children. A qualitative method of differentiating these children, based not only on their response to sound but also on a careful analysis of their total behavior pattern, has been employed in a group study of 60 children at the Children's Memorial Hospital. The lack of normal speech was found to be due to deafness or impaired hearing in 50 per cent, and to other disabilities in the remaining 50 per cent of these children. A preliminary report on this study is herewith presented.

Hearing and Speech

Although deafness may be suspected early in an infant's life it is usually not until the child has failed to learn speech by the age of two or two-and-a-half years that the parents seek medical advice. A child may have other obvious disabilities, but the lack of speech immediately raises the question of whether or not the hearing is normal.

Normal Speech

Assuming the presence of normal speech organs, the development of speech depends upon a normal peripheral auditory mechanism, with properly functioning connections, *1*—to the

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auditory centers in the cortex (see Fig. 1). *2*—to the four sensory and motor speech areas, namely the frontal, temporal, parietal and supplementary areas which are usually present only on the left side of the brain (see Fig. 2), and *3*—to the centers for vocalization and articulation which are located on both sides of the brain (see Fig. 3). The connections between the auditory, speech, motor and other cortical areas

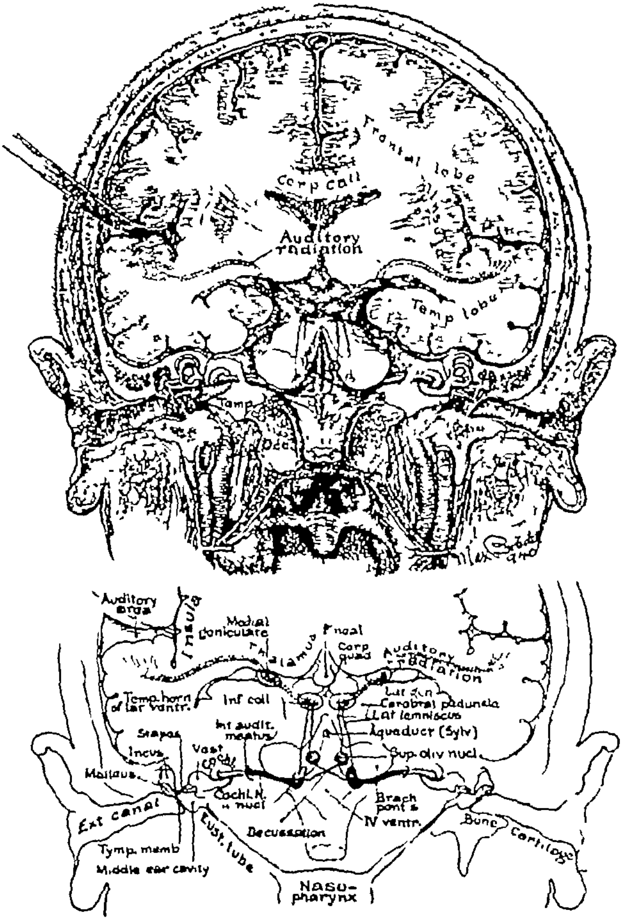


Fig. 1. From the Otological Research Laboratory and the Department of Art in Medicine, Johns Hopkins University, by the late Max Brödel (1940 Year Book of the Eye, Ear, Nose and Throat).

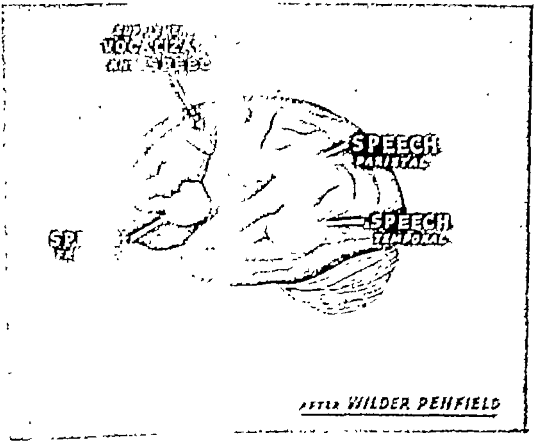
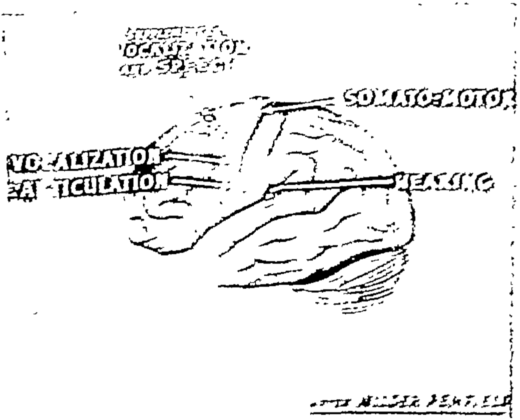


Fig. After a drawing L. Roberts (Localization of Speech in the Cerebr Cortex) 1951



which are necessary for the reception, understanding and reproduction of the spoken word are all presumed to be subcortical (see Fig. 4).

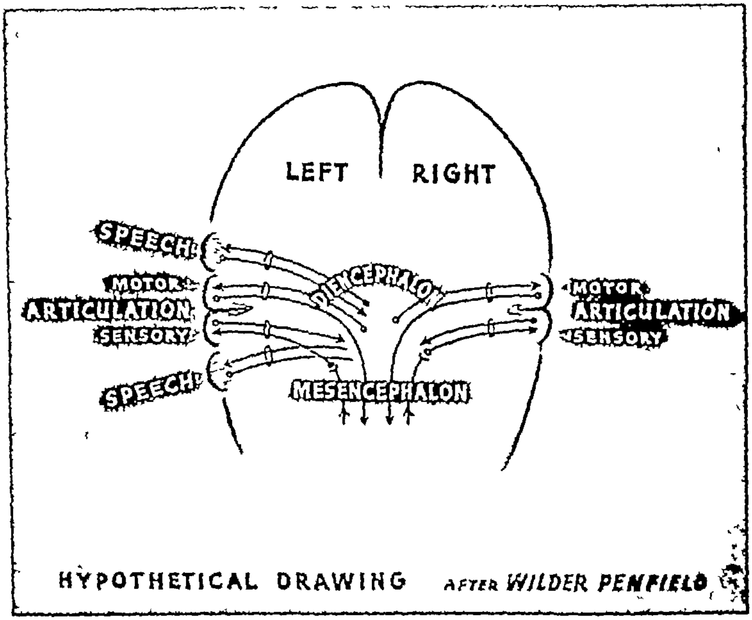


Fig. 4. Hypothetical diagram from "The Cerebral Cortex of Man," by Penfield and Rasmussen, 1950.

Lack of Normal Speech

Normal speech development, therefore, may be delayed or prevented by, *1*—a lesion in the peripheral auditory apparatus, such as congenital or acquired atresia of the external canal, with or without malformation of the ossicular chain, or disease in the middle ear, which would result in a conductive hearing loss; *2*—developmental anomalies or diseases affecting the organ of Corti in the cochlear duct, the ganglion cells, or the auditory nerve itself, which would produce a perceptive hearing loss, or complete deafness. The vestibular apparatus may, or may not, be involved in any of these conditions.

Failure of normal speech development may also be due to some central dysfunction, cortical or subcortical, with motor or pyramidal tract involvement, as in cerebral palsy, or without motor involvement. In either case damage may involve one or more of the speech areas, resulting in motor or sensory aphasia. Minimal damage or dysfunction in the subcortical areas could also conceivably result in delayed or absent speech development.

Behavior of Handicapped Children

In considering children with speech disturbances it has been recommended by Myklebust that "rather than assess only the child's response to sound, the total behavioral pattern should be analyzed." He adds that "this approach has the advantage of recognizing that a certain child may not respond to sound for other reasons (than deafness), such as marked psychic disturbances, or he may respond inconsistently because of perceptual problems associated with brain injury." He emphasized that "the diagnostically differentiating characteristics should be those other than the ability or capacity to respond to sound." Hirsch stated that "the loss of hearing has ramifications that go beyond the mere loss of communicative skill to basic changes in the personality." The behavior of any child is the result of many factors, but the behavior pattern of the deaf child is basically the result of his disability, providing his disability is only the absence of hearing. Brain injured children show behavior disorders and perceptual problems peculiar to their disability, as described by Strauss and Lehtinen, while emotionally disturbed children may exhibit the peculiarities of infantile autism or schizophrenia described by Kanner. Many of the aforementioned children have been confused in the past with the mentally retarded. It would appear, therefore, that the behavior of these handicapped children is, at least in part, an expression of their disability or disabilities. There are cases that exhibit the features of two or more of these groups and these are the most difficult to differentiate at a time when a correct diagnosis is important for their rehabilitation.

Purpose of Study

The purpose of this study has been to apply the method established by Prof. H. R. Myklebust, and used in the Children's Hearing and Aphasia Clinic at Northwestern University, to determine the major cause for the lack of normal speech in this group of 60 children.

Principles of Method

The basic principle of this method is to apply to the clinical practice of pediatric audiology that knowledge which is pertinent from the fields of psychology, psychiatry and special education. This method of investigation provides a realistic appraisal of the whole child. It recognizes the special problem of multiple handicaps, *i.e.*, impaired hearing and cerebral palsy, and it suggests that the child with more than one disability is best classified, for purposes of education and rehabilitation, on the basis of his major handicap.

Method Employed at Children's Memorial Hospital

The principles of this technique have been employed and expanded in this study over the past three years. The program at the Children's Memorial Hospital is primarily an inter-professional group study of each child. Information relative to every aspect of the child's problem is obtained from the detailed history, through ear, nose and throat examination, hearing tests, psychometric and speech evaluations. An electro-encephalogram has been obtained on each of these children, for evidence of focal or diffuse irregularities. When indicated, special blood studies, X-rays and consultations have been obtained with Psychiatry, Neurology, Ophthalmology and Pediatrics to make the investigation as complete as possible.

Steps in Differential Diagnosis

The first step in the differential diagnosis is a very complete history. This is obtained from the answers of the parents to a questionnaire similar to that prepared and used by Myklebust. This consists of a list of specific questions designed to obtain all possible information of etiological and diagnostic significance. This includes details of pre-natal, birth and postnatal conditions, history of all illnesses, operations and injuries, genetic development, emotional adjustment, auditory and language behavior. This information provides important differential clues as to whether the problem is organic or functional and, if organic, whether it is peripheral or central. This data is obtained by the otologist before the child is examined or the hearing capacity tested, so that the hearing tests may be modified to conform with the type and age of the child under investigation. Valuable impressions may be gained if the child's behavior is observed during the history-taking period.

The second step is a thorough examination of the ears, nose and throat for evidence of disease or any condition which would affect the hearing, speech, or general health of the child. The integrity of the vestibular portion of the inner ear may be determined by caloric or rotation tests. This is important in all cases of suspected total deafness, in one or both ears.

In spite of the variety of methods used for testing the hearing of infants and pre-school children, accurate measurement of their hearing capacity still remains a difficult and challenging problem. Clinical experiences suggests, *1*—that the application of adult hearing test techniques to these young children is unsatisfactory, *2*—that pure tones may be too abstract and meaningless as auditory stimuli to brain injured children with perceptual disturbances, and *3*—that the child who fails to respond to sound immediately is not necessarily deaf.

The third and fourth steps in the differential diagnosis are combined. The hearing tests have been carried out in a sound-treated room with the child seated before a small table. While engaged in play with simple "educational" toys, many features of his behavior may be observed and noted; his general adaptability and his attitude toward strangers; his response to simple instructions by gesture or unexpectedly to verbal commands; his preference for the right or left hand; his ability to grasp and manipulate various objects, build simple structures, etc.; in brief, his general physicial and motor development, sensory and perceptual alertness, vocalizing ability, and emotional stability may be evaluated. The information obtained from the answers to the questionnaire may thus be correlated with the clinical study of the child during the hearing test period.

Behavior Characteristics

Inasmuch as the basis of this method is to assess, not only the child's response to sound but also his total behavior pattern, it is necessary at this point to outline briefly the characteristics of each group. It must be emphasized, however, that there are many variables. Some children do not fit exactly into a specific pattern, often because they have multiple handicaps; nevertheless, there is a pattern of general behavior which is recognizable for each of these groups: the deaf, the brain injured, the autistic, and the mentally retarded.

The Deaf Child

The behavior of a deaf child is different from that of a child with normal hearing. The loss of the primary warning sense, hearing, increases the dependence on the other special senses; therefore, the deaf child is especially alert to vibrations and visible movements in the environment. Unable to hear and speak, he communicates and makes his wants known by using gestures and pantomime, because he has good inner langauge and understanding. Failure to make himself understood may give rise to frustration tantrums. His voice tends to be flat and toneless. He does not improvise or play with sounds. His motor development is good, and he tends to be noisy and hyper-active. The deaf child is in general intelligent, friendly and cooperative at this age. He is generally consistent in his day to day behavior and not too difficult to manage in the home, providing the parents are intelligent, understanding and affectionate in the handling of his problem. If he is not totally deaf he will respond to sound, like a normal child, when the intensity reaches his threshold of hearing. This pattern of behavior is characteristic of the congenitally deaf child. It has been used in this study as a frame of reference for those who are presumed to be deaf.

The Brain Injured Child

The brain injured child is different in every respect. As a result of the brain damage caused by trauma, toxins or infection before, during or after birth, he may have disturbances of perception, concept formation, language and emotional control which are reflected in his behavior (Strauss and Lehtinen). These subtle expressions of brain injury may or may not be associated with the type of motor involvement seen in cerebral palsy. From our present point of view his basic disability is abnormal perception. He may misidentify visual, auditory, tactile and kinesthetic stimuli. Because all stimuli have equal power to attract him, he is distractible, hyperactive and inconsistent. Because the usual control, or inhibiting effect, exercised by the higher centers of the brain over the thalamus and hypothalamus is lacking, his emotional behavior is unstable and exaggerated. He is disinhibited, fearless, happy and affectionate. He is given to violent, uncontrollable outbursts of temper. He is destructive without anger. He is meticulous and pays unusual attention to minute details. He may be very friendly and intelligent, but he is inconsistent in his day to day behavior. He has a tendency to persist in repeating an activity once begun, long after it has ceased to have any meaning or purpose. This characteristic is called perseveration. He is difficult to understand and manage in the home, because he is so unpredictable and disorganized. Although he may be presented as a retarded child or behavior problem, his disturbed language development raises the question of whether or not his hearing is normal. His response to sound is inconsistent and at times remarkable. He may not respond to pure tones presented in the free field at intensities approaching the threshold of pain, and yet a moment later definitely respond to a whispered voice or the squeak of a toy rubber mouse, depending upon his attention at that moment.

The Autistic Child

The autistic child is very strange. He may be confused with the feeble-minded or, because of his muteness and inadequate responses to sound, with the deaf. The behavior characteristics of this severe form of psychic disturbance were first described by Kanner in 1943 and called infantile autism. This type of child is withdrawn, non-communicative, self-sufficient, happiest when left alone, meticulous in details, adverse to changes in routine, interested primarily in objects and not people, not even his own parents. He treats people as objects, shows no affection, and will rarely look anyone in the face. These children are intelligent, but their "cognitive potentialities are masked by their basic disorder," which has been defined as "a disability to relate themselves in the ordinary way to people and situations, from the beginning of life." The child's behavior is "governed by an anxious obsessive desire for the maintenance of sameness that nobody but the child may disrupt" (Kanner). He is given to panic tantrums, followed by sudden calm. His face is placid, but occasionally he may be found smiling, humming or singing to himself. Hebephrenic and catatonic types of schizophrenic behavior may also be observed in the most seriously disturbed children. The hebephrenic type is characterized by curious mannerisms, gesticulations, tic-like movements, and unexpected spells of laughing and crying. Compulsive and repetitive actions are common. The catatonic type is more generally tense, rigid, and contrary. Awkward positions may be maintained for long periods. The autistic child pays no attention to his surroundings. He may not respond even to a painful stimulus. He may appear to be in a stupor. Here, again, it is the lack of speech, and absent or inadequate response to sound, which may lead to a suspicion of deafness. The child's characteristic pattern of behavior, however, provides a clue to the basic disability. An accurate assessment of the hearing capacity is most difficult because of the child's self-imposed isolation and lack of normal response to any stimulus. The pathology of this condition is unknown. Although some of these children may remain mute, the majority apparently have normal hearing.

The Mentally Retarded Child

The mentally retarded child performs at the level of his intellectual or mental maturation. He is retarded in all phases of his development, including language and perception. He is unable to take care of himself. His social, learning, and mental age do not relate favorably to his chronological age. The various types of idiots, morons, feeble-minded and so-called inadequate children can be differentiated by specific tests which are best applied by those who are qualified, by special training, to interpret the results in terms of educability of the child in question. If there is a suspicion of a hearing impairment, the type of hearing test employed must be in keeping with the mental age of the patient under consideration.

Objective of Hearing Tests

The primary objective of these hearing tests has been to determine whether or not the child who lacks normal speech has adequate hearing to learn speech. For this purpose it is sufficient to determine that his unaided hearing is within the zero to 30 decibel zone for the speech frequencies.

Hearing Test Equipment

The hearing test equipment includes (1) noise makers, such as tin crickets, rubber mice, sleigh bells, rattles, whistles, etc., and (2) children's rhythm band percussion instruments, including a metal triangle, cymbal, "A" chime, tambourine, wooden tone blocks and castanets. The intensities (at one and three feet) and the frequency range of all these have been determined as accurately as possible at the National Research Council Laboratories, Ottawa. With an Octave Band Pass Filter these "calibrated" noise makers were found to have peak frequency ranges as follows: tambourine, 200 to 800 cycles per second when the drum part was tapped, and 3,200 to 6,400 when the instrument was shaken; wooden tone block, 800 to 1,600 cps.; large cricket, 1,600 to 2,400 cps.; small tin cricket, 3,200 to 6,400 cps.; sleigh bells, 2,400 to 9,600 cps., and a squeaking rubber mouse, 6,400 to 8,600 cps. The intensities range from 20 to 100 decibels at one foot. (3 Pure tones from an audiometer are presented through a loudspeaker situated three feet away from the child. (4) Recorded "familiar" sounds are also presented through the loudspeaker into the sound field. These include the sounds of a train whistle, vacuum cleaner, telephone, aeroplane, auto horn, cat, dog, cow, bird, bell, etc., and the voices of a woman, man and child. All of these may be presented at controlled intensities. (5) Simplified live voice tests may be used, when indicated, to determine a child's capacity to hear and understand speech. An approximate speech reception threshold may thus be obtained. (6) Recorded music has been employed in cases of suspected aphasia when the responses to the other tests were inconsistent but suggested that the hearing was adequate to learn speech. In several such cases the child danced to the music when it was presented at an intensity level well within the limits of normal hearing. This response was repeatable and, as far as could be determined, not due to the feeling of vibrations.

The child's vocalizations provide valuable information. If an infant will imitate humming sounds, or repeat baa, baa, mom, mom, after the examiner, there is definite evidence that he heard these sounds. This vocalization test is very useful for the brain injured or mentally retarded child, to demonstrate that he responds at a conversational level.

Hearing Test Technique and Response

As mentioned before, these objective hearing tests have been carried out in a sound treated room, with the child seated at a small table. While an assistant keeps the child occupied in a play situation with toys, the examiner activates the calibrated noise makers at a distance of three feet behind the patient, beginning with those of the lowest intensity. If no response is obtained, the tests are repeated at a distance of one foot behind the patient, or at either ear. Although great care is taken to keep the noise makers out of sight during these tests, they may be brought into the periphery of the child's visual field deliberately to note his reaction. Deaf children are usually very alert to movements in their environment, whereas children with severe psychic disturbances may completely ignore the sound makers, even when the instruments are struck directly in front of them. Pure tones and recorded familiar sounds are then presented in the free field at increasing intensities. In our experience, children of the pre-school age group do not tolerate head phones very well. hence the almost exclusive use of the free, or sound, field method. The most commonly observed response to these auditory stimuli is cessation of activity, with head turning to identify the source of the sound. A startle response, reflex eye blinking, or eye turning may be observed when loud sounds are presented to young infants. It is noteworthy that once a response is obtained the child, even with normal hearing, may not respond again until a different or stronger sound stimulus is presented.

It is very difficult to assess accurately the hearing of brain injured children with the type of motor involvement seen in cerebral palsy. They have to be "structured" in their hearing tests by eliminating all other stimuli and by having them visually identify and play with the sound makers before they are presented as auditory stimuli. In this way responses may be obtained in otherwise apparently deaf children.

Interpretation of Responses

The child's response to sound is interpreted in relation to the behavior pattern observed during the hearing tests. The history, physical examination and clinical analysis of the behavior pattern are thus correlated and used to validate the responses, or lack of responses, to the hearing tests. The technique of applying these hearing tests has been standardized, and an approximate audiogram has been obtained on all of the children in this study.

Classification of Hearing Test Results

Regardless of the child's major handicap, his hearing has been classified as follows:

1. *Adequate to learn speech,* when he responds to the auditory stimuli mentioned above, at intensities of 30 decibels or less, in the speech frequencies.

2. *Impaired hearing for speech development,* when he responds to stimuli at intensities of not less than 35 to 65 decibels. This degree of hearing loss is usually due to a conductive lesion, and often it can be raised to a useful level by amplification or treatment.

3. *Inadequate to learn normal speech,* when he fails to respond to stimuli of less than 70 decibels. This is most commonly due to an inner ear or perceptive lesion, with or without a conductive component.

4. *Deaf,* when he fails to respond to any of the sound stimuli.

This classification may then be used as a guide in planning the child's medical and educational rehabilitation program.

Correct Diagnosis

In evaluating a child suspected of having impaired hearing by this qualitative method, the diagnosis is based on the history, physical examination, hearing assessment and analysis of the behavior pattern. This method has proven to be practical in differentiating the deaf child from those children whose lack of speech is due to some form of central damage or dysfunction.

A correct diagnosis is obviously necessary for the proper education, support and guidance of the parents of these children. They need assistance and encouragement in the understanding and handling of their problem in the home.

The necessity for a correct diagnosis is emphasized in outlining a program of medical, psychological and specialized educational rehabilitation for each of these exceptional children, since each of the four groups mentioned requires a different type of rehabilitation program.

RESULTS.

The average age at which these children were first examined was 31 months. They were all suspected of having impaired hearing because of their speech deficiencies. It was found that 25, or 42 per cent of this group suffered multiple handicaps. Classified on the basis of their major handicap, for purposes of medical and educational rehabilitation, 30, or 50 per cent, had insufficient hearing to learn speech because of some peripheral auditory disturbance. Of these, 14 were deaf, 14 had a very severe hearing loss (of 80 decibels or more), and two had a severe hearing loss (of 60 to 80 decibels).

The remaining 50 per cent were considered to have adequate hearing to learn speech. Their lack of normal speech was apparently due to:

1. Brain injury in 29 per cent of the total 60 children. One-third of these children had cerebral palsy. The diagnosis of brain injury was confirmed in the majority of the remaining cases by an abnormal electroencephalogram.

2. Mental retardation in 13 per cent of the total. The degree of retardation was established by the psychometric evaluation.

3. Severe emotional disturbances in 8 per cent of the total e.g., autism and schizophrenia. This diagnosis was confirmed by the psychiatric consultant.

CONCLUSIONS.

1. The purpose of this study has been to evaluate the method of investigation recommended by Myklebust for children who are suspected of having impaired hearing because of their lack of normal speech.

2. This method has been employed and expanded at the Children's Memorial Hospital by organizing an inter-professional diagnostic group. This consists of representatives from otology, audiology, speech therapy, psychology, psychiatry, neurology, pediatrics, ophthalmology, electroencephalography, and social service. The specialized knowledge of these consultants has been applied to the final assessment of each of these handicapped children, and utilized in proposing a program of rehabilitation.

3. This qualitative method has proven to be practical in differentiating the deaf child from those children whose lack of speech is due to some form of central damage or dysfunction.

4. Although the elaborate plan of total investigation described in this paper was organized for a specific purpose, the principles of this method may be applied by the otologist as a valuable diagnostic guide in clinical practice. He may thus be assisted in planning the correct program of investigation or rehabilitation of these handicapped children.

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I CONGRESO EXTRAORDINARIO DE LA SOCIEDAD INTERNACIONAL DE AUDIOLOGIA.

Buenos Aires - Republica Argentina.

The First Extraordinary Congress of the International Society of Audiology, with headquarters in Paris, will be held in Buenos Aires, November 26 - 30, 1954.

This important scientific gathering held outside France for the first time, will be honored with the presence of prominent international scientists, specially invited, from the United States, France, Belgium, Switzerland, Holland, Britain, Germany, Brazil, Chile and Paraguay. It has also been supported by all the Universities of the country, of well known local and foreign centers of studies, as well as of delegates from several European and American countries.

For further information address Dr. Atilio Viale del Carril, or Dr. Renato Segre, general secretaries, Buenos Aires, Argentina.

LARYNGEAL COMPLICATIONS OF ENDOTRACHEAL ANESTHESIA.*

WALTER H. MALONEY, M.D.,
Philadelphia, Pa.

It is generally recognized that endotracheal intubation has now assumed a permanent place in anesthesia. Because of the universal use of this method of administering anesthesia it is important to recognize the accompanying hazards and contra-indications to such a procedure. Such recognition will, by our awareness of them, eliminate many of the problems. This paper discusses the basic etiologic factors, diagnosis, and treatment of these complications.

The careful observer can elicit symptoms and see objective findings indicating laryngeal pathology in as high as 50 per cent of the cases in which endotracheal intubation was performed if he records all minutia; however, there is a relatively low incidence of persisting serious pathology requiring active treatment. It is important to remember that serious complications do occur and must be recognized early to prevent disability or even death. The specific problems with which we must concern ourselves range from submucosal hemorrhage, abrasions of mucous membranes, contact ulcers and granuloma, membrane formations, subglottic edema, lacerations, to perforations of the tracheo-bronchial tree or esophagus.

ETIOLOGY.

The etiologic factors may be divided into two parts: the first, the intrinsic factor, refers mainly to the normal anatomy of the hypopharynx, larynx, and trachea. The anatomy is such that we can anticipate the points of stress and trauma. The action of the larynx is to protect the tracheo-bronchial tree as well as to act as an organ of phonation, and as such, it has a powerful sphincteric reflex which is activated at the slight-

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* Read before the Seventh Annual Post-Graduate Course on Diseases of the Chest, sponsored by the American College of Chest Physicians and Laennec Society of Philadelphia, March, 1954.

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est stimulus. This results in considerable trauma to the intrinsic larynx during severe cough and spasm. There is a constant motion of the arytenoids with respiration which results in continuous trauma to the cords, even under anesthesia, should a foreign body such as an endotracheal tube be in place. The vocal process of the arytenoid projects into the vocal cord and is covered only by a thin layer of perichondrium and mucosa. This makes it extremely responsive to trauma.[1] The pyriform sinuses with the cricopharyngeus muscle guiding the entrance to the cervical esophagus are easily accessible to perforation. The laryngeal ventricles are so placed that the endotracheal tube will enter into the ventricle rather than the trachea should the bevel be turned to the side or the cords be in spasm. The subglottic area with its loose areolar tissue quickly becomes edematous after trauma. All of these are normal anatomical facts.

We must also keep in mind the abnormalities of these structures due to super-imposed intrinsic factors. This includes post-irradiation edema and dryness of the mucous membrane. Also chronic infection of the mucosa results in a loss of ciliary activity, increased friability, and desquamation of the mucous membrane. All of these add up to an increased sensitivity to trauma.

When considering the extrinsic factors in the etiology of laryngeal complications, number one on the list must be the technique of the operator. Long ago, Dr. Chevalier Jackson described the proper technique for intubation of the larynx.[2] It has not changed over the years. In endoscopy there is no place for the heavy hand. It will be agreed, I am sure, that direct vision has an advantage over blind intubation in avoiding trauma. There must be a careful choice of instruments insofar as size and condition are concerned. This will be discussed more fully later. The proper plane of anesthesia must be obtained to avoid spasm and resistance to the passage of the tube. It is important to maintain this proper plane of anesthesia to avoid coughing, swallowing and other factors causing continued motion of the tube or of the larynx on the tube. Finally, it is important to avoid makeshift apparatus.

DIAGNOSIS.

The diagnosis of laryngeal complications is made on the evaluation of the patient's complaint plus direct examination and objective findings as shown by the laryngeal mirror or laryngoscope.

Laryngeal complications can be divided into two basic groups: there are those which occur immediately or within the first 48 hours. These include abrasions, submucosal hemorrhage, edema, laceration, and membrane formation; then, there are those which may develop from a few days to six months later, such as ulceration and granuloma formation. All of these patients will complain of a sense of discomfort in the throat which may become severe enough to be described as actual pain. Hoarseness is also present in nearly all cases.[3] These complaints, however, when associated with progressive stridor, dyspnea, restlessness, anxiety, tachycardia, sternal retraction, and finally anoxia and exhaustion, call attention immediately to an obstructive phenomenon in the upper respiratory tract. In nearly all cases this will be due to laryngeal edema or to the formation of a subglottic membrane.[4] While they occur most frequently in infants and children, they can also occur in adults. The presence of a subcutaneous emphysema is indicative of a perforation. Severe dysphagia may be associated with perforation but is also present following traumatic laryngoscopy. X-rays, of course, are important for those cases of suspected perforation. In the last analysis, the diagnosis of laryngeal pathology is confirmed by mirror laryngoscopy and/or direct laryngoscope. Treatment should be instituted immediately as indicated. Care must be taken during mirror laryngoscopy to avoid missing the subglottic stenosis or obstruction since the supraglottic structures may be normal in appearance. Negative mirror laryngoscopy, in the presence of positive signs and symptoms, indicates the need for direct laryngoscopy, tracheoscopy, or bronchoscopy.[5]

TREATMENT.

Treatment must be divided into two phases: the first is the *prophylactic regime* which is carried out both by the anesthesiologist and by the attending surgeon or physician. Preoperative elimination of chronic infection in the sinues, oral cavity and pharynx and tracheo-bronchial tree by chemotherapy is valuable. Again we must reiterate the need for gentle, atraumatic, carefully executed intubation with proper instruments. Not only is the proper size of the instrument important, but cleanliness is also a factor too often neglected. Introduction of a virulent organism into an abrased mucous membrane is not to the best interest of the patient. Endotracheal tubes must not be allowed to become roughened by careless handling. Stylets are dangerous, and care must be used to avoid trauma to the anterior wall of the trachea or pyriform sinus. The lubricating agent may be irritating and cause a tissue reaction. Care should be taken to place the cuff well below the larynx, keeping it small and the surface smooth. Any tube which gives an air tight fit in the trachea without inflating the cuff is one which is liable to cause a subsequent edema.

The use of endotracheal anesthesia in children should be a matter of consultation between the surgeon and the anesthesiologist. We know that these are the cases where it is difficult to predict the reaction of the larynx to intubation; therefore, it would seem logical to avoid endotracheal anesthesia in such patients except where there is a definite need for it. The anesthesiologist should not become so dependent upon the tube that he fails to develop his own full scope of anesthetic techniques and thus be forced to use the endotracheal technique where it is not absolutely necessary.

Inhalation therapy, humidity, systemic chemotherapy, vocal rest, and avoidance of irritants such as talking and smoking are helpful in preventing further reaction to a possible traumatized larynx in the postoperative care of the patient following endotracheal anesthesia.

Direct treatment of these complications refers in the main to the endoscopic removal of granuloma or of the fibrinous exudate accumulating in the subglottic space, as well as to tracheotomy. While we try to avoid the need for direct treatment in a way described previously, should mirror or direct laryngoscopy show the formation of a granuloma or fibrinous exudate, direct intervention is indicated. The granuloma of the vocal cords usually occurs in the posterior third of the cords and is often bilateral. This will often recur following direct laryngoscopic removal, and it has been found that the smaller the stalk of the granuloma, the less chance of recurrence. An occasional granuloma may become pedunculated to the point where it will be extruded by a sudden cough. This does not happen too frequently and one should not rely upon this method of treatment. Direct laryngoscopy with removal of the excess tissue followed by a strict regime of vocal rest and elimination of irritants is the indicated treatment when the diagnosis of contact ulcer granuloma is made. Where there is no granuloma, the contact ulcer will usually heal on a regime of vocal rest and speech therapy.[6] Of course, severe edema with obstruction of the airway may result in the need for tracheotomy. Be sure that it is true edema and not a membrane which can be removed by direct laryngoscopy. Tracheotomy should be carried out early to avoid fatality.[7] There is no question that in this instance procrastination is dangerous. The safe rule to follow is that when one begins to debate the need for tracheotomy, then is the time to do it.

Finally, a word about division of responsibility in these problems. There must be a close coordination between the laryngologists and the anesthesiologists. Responsibility must be clearly defined. The person called for the emergency consultation must be equipped to carry through the immediate examination and treatment; that is, mirror laryngoscopy, direct laryngoscopy, intubation, and tracheotomy. Proper equipment must be available for use in such emergencies. There is a point in the examination of these patients, particularly in children with obstructive membrane formation or edema, where one must proceed with the removal of the membrane or insertion of an endotrachial tube or bronchoscope, or the resultant spasm and reduction in airway may result in asphyxia of the patient. It is dangerous to take a look at a larynx with a direct laryngoscope in patients with obstructive phenomenon without having available the endoscopic or tracheotomy equipment to re-establish the airway quickly.

CONCLUSION.

I have discussed the underlying factors in the etiology, diagnosis, and treatment of laryngeal complications of endotracheal anesthesia. We must make every effort to reduce them. Endotracheal anesthesia is here because there is a real need for it. We must progress in turn and establish the techniques which will reduce these complications to a negligible degree. This can be done by proper attention to the basic concepts of anatomy, physiology, and endoscopic technique.

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230 North Broad Street.

BRONCHOESOPHAGOLOGY COURSE.

The next Bronchoesophagology Course to be given by the University of Illinois College of Medicine is scheduled for November 8 - 20, 1954. Under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

COTTON GRANULOMA OF THE MASTOID.*

CASE REPORT.

RICHARD J. CROSS, Major, USAF (MC) and
WALTER JORDAN BROWER, Captain, USAF (MC),
Baltimore, Md.

The following case presents a most interesting and unusual aspect of chronic middle ear disease.

HISTORY.

The patient, J. R. G., a 69-year-old white male, dependent of an officer, was seen by the Department of Otolaryngology at the School of Aviation Medicine in November, 1952, with the complaint of vertigo, tinnitus, headaches, cervical pain, and a discharging left ear. His difficulty began when he was six years old. Following an attack of measles the patient developed bilateral acute suppurative otitis media. He recovered from the measles, but the ears continued to discharge, and this condition progressed into chronic suppurative otitis media. The patient consulted an otologist who treated and eventually cleared up the discharge from the right ear; however, the left ear did not respond to treatment and continued to suppurate throughout his adult life. At the age of 37, the patient was advised to undergo a mastoidectomy as a possible means of stopping the discharge. He consented and a simple mastoidectomy was performed on the left ear at that time. Postoperatively, the patient's condition improved to the extent that the amount of discharge diminished but did not cease. Five years after the operation the patient experienced severe attacks of vertigo followed by nausea. He sought the advice of a gastroenterologist who, after an examination, suggested that he consult an otologist concerning his ear. The otologist instructed the patient to keep the left ear clean and dry. Since that time the patient has meticulously cleaned his left ear with a dry cotton swab every day. In 1949, the patient was hospitalized because of a genito-urinary complaint and, while in the hospital, had an otolaryngologic consultation because of his chronically draining left ear. Examination at that time disclosed a large polypoid mass in the left middle ear. This was removed by means of a snare through the external canal. Microscopic studies of this tissue revealed it to be a polyp with excessive fibrosis. The patient was somewhat improved after this procedure, except for the persistent discharge and occasional attacks of tinnitus and vertigo. For the past six months he has been suffering from generalized headaches and cervical pain, with spasm and twitching of the left side of the face. His hearing has been impaired in both ears since youth, and he has worn a bone conduction type hearing aid on the right ear for 35 years.

Past History:

The patient had a tonsillectomy and an adenoidectomy when a child. A submucous resection of the nasal septum was performed when he was 32 years old. He has no known allergies or sinusitis.

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* From the Department of Otolaryngology, USAF School of Aviation Medicine and Department of Radiology, Randolph Air Force Base Hospital, Randolph Field, Texas.

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EXAMINATION.

Examination of the right ear revealed the external canal to be normal. The tympanic membrane was scarred, retracted and distorted. There appeared to be a small perforation in the anterior, inferior quadrant, in which a slight amount of mucoid discharge was present. Upon examination of the left ear a well healed postauricular scar and a slight pit-like depression in the left mastoid process was noted. The external canal was normal. The tympanic membrane was absent. There was a large pulpy mass that filled the middle ear cavity and extended to the level of the tympanic ring. Purulent material was observed around this tumor-like mass. A biopsy of the mass was taken. The nose, throat and nasopharynx were essentially negative. Bone conduction was good in the right ear and fair in the left. Air conduction was poor in both ears. Roentgenograms of the temporal bones (see Fig. 1) revealed the

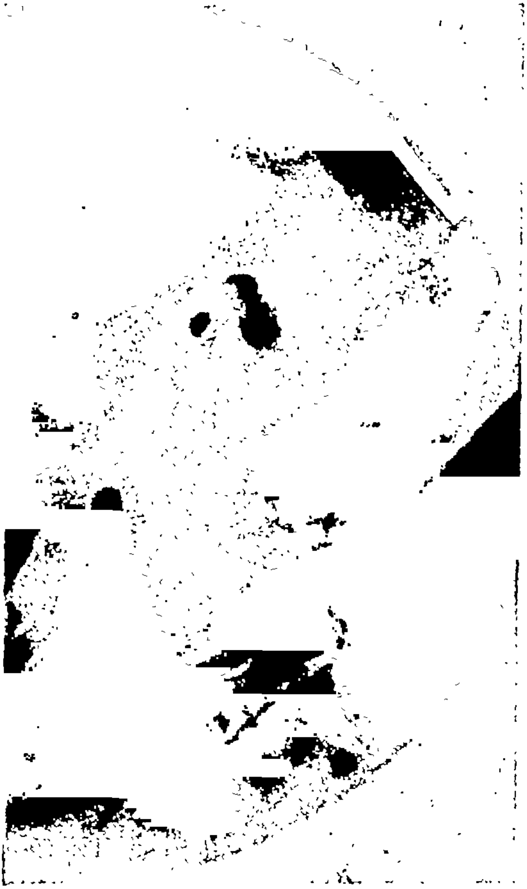

Fig. 1. Lateral view of the left mastoid. A large and fairly distinct area of erosion is evident adjacent and posterior to the canal.

presence of a few air cells in the right mastoid amid considerable sclerosis. The few cells present were fairly distinct, and there was no suggestion of active inflammation. On the left there was a large punched-out defect posterior to the auditory meatus which had sharp, well-defined edges. No definite air cells could be demonstrated. The mastoid process was found to be grossly sclerotic and in addition showed evidence of an old mastoidectomy. The roentgenographic evidence of erosion suggested the presence of a neoplasm. possibly arising in an area of squamous metaplasia. Cholesteatoma was also considered, but the edges of the eroded area were not as ragged and indistinct as they might be if the bone were being destroyed by a malignant neoplasm or destructive mastoiditis. Abscess formation is less common than cholesteatoma in the sclerotic mastoid.

The pathologic report of the biopsy of the mass in the left ear revealed it to be a granuloma resulting from chronic inflammation. There was no evidence of malignancy present. The impression was: *1.* Pyogenic granuloma in the left middle ear and attic which produced erosion in the attic, secondary to chronic suppurative otitis media; and 2. Chronic suppurative otitis media, right, quiescent. Because of the evidence of erosion in the left attic the patient was advised to undergo a radical mastoidectomy. This was performed on February 3, 1953.

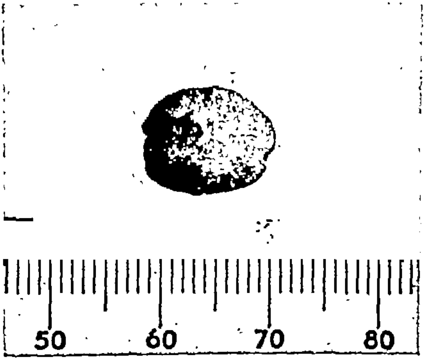

Fig. 2. Cotton foreign body removed from the mastoid antrum.

OPERATION.

Under general anesthesia the left auricular area was prepared in the routine manner. A postauricular approach was used. The skin and subcutaneous tissue were incised and retracted, exposing the mastoid process of the temporal bone. Upon examination a large eroded area of bone approximately 1 cm. in diameter was found in the region of the antrum. This eroded area was covered only by periosteum. The periosteum was incised and retracted, and a large amount of granulation tissue and debris was noted in the antrum. This was removed by curettage and inspection revealed a hard, fibrous-looking mass that appeared to be firmly imbedded in the antrum. After careful dissection this mass was removed in its entirety and found to be an old piece of cotton (see Fig. 2) that persumably had been left in the antrum at the time of the previous surgery over 30 years ago. This cotton measured 9 x 12 mm. Exploration of the attic disclosed erosion through the tegmen, so that a small area of the dura was visible. The erosion also extended through the facial canal and exposed the VIIth Nerve. Granulation tissue and necrotic material were curetted from the middle ear and the attic. Rough and overlying bone was smoothed and removed with a gouge and curette; the tympanum, malleus and incus were not found. The mastoid cavity was developed in the routine manner. Because of the patient's age, extensive dissection was not attempted and only a modified radical mastoidectomy was performed. A T-shaped plastic flap was made in the external canal to line the mastoid cavity. The incision was closed and a compression dressing was applied. The patient withstood the procedure well and was returned to his room in good condition.

Postoperative Period:

Recovery was uneventful. The patient was up, and sitting by his bed on the first day. There was a large amount of sero-sanguineous discharge for several days. The dressings were changed daily; the drain was shortened every other day and finally removed completely on the ninth postoperative day. The sutures were removed on the sixth day and the postauricular incision was well healed. From the ninth to the 16th day the opening in the external canal was inspected and cleansed of debris and discharge by means of suction. On the 20th day the ear was almost completely dry, and examination revealed a clean, well healing cavity. The patient said that he felt improved and had not had an attack of vertigo or tinnitus since the operation. Roentgenograms of the mastoids 21 days after the operation showed that the large defect in the left mastoid had filled in considerably. A portion of the bone was missing posteriorly following surgery, but apparently the area of bone destruction was regenerating with new bone following removal of the foreign body.

A communication from the patient in November, 1953, approximately one year after the operation, related that he was asymptomatic and in good health despite his age.

DISCUSSION.

This case clinically suggested an expanding lesion of the mastoid antrum which was producing pressure necrosis. Roentgenologically it did not resemble a cholesteatoma. The cotton foreign body had been present in the mastoid antrum for 32 years and probably was the reason why the original operation did not give the expected result. This foreign body was firmly entrenched in the antrum and could have reached that point only through a previous operation. It would obviously have been impossible for the patient to accidentally push the cotton into this area while cleaning his ear.

The area of erosion in the antrum was larger than the cotton foreign body. It is doubtful whether the foreign body alone could have produced this result. The role of secondary infection and granulation tissue formation is an important one. The fact that the patient lived for 32 years with only occasional difficulty indicates the slow process involved.

CONCLUSIONS.

1. This case presents an unusual aspect of chronic middle ear disease and illustrates the importance of careful inspection of the mastoid cavity and surrounding structures before completion of any type of mastoid surgery.

2. Radiologically the erosion in the mastoid antrum was not typical of cholesteatoma, neoplasm or mastoiditis, although it did resemble a tumor to some extent.

3. It is felt that the erosion was caused by a combination of the cotton foreign body, secondary infection and granulation tissue rather than by pressure of the cotton alone.

104 W. Madison Street.

ANNOUNCEMENT.

An introductory course in Reconstructive Surgery of the Septum and External Nasal Pyramid will be given, under the direction of Dr. Maurice Cottle, March 19 through 26, 1955, at the University of Oregon Medical School. This will be sponsored by the Department of Otolaryngology.

BOOK REVIEW.

Anatomy of the Bronchovascular System. Its Application to Surgery. By George L. Birnhaum, M.D., Chief, Surgical Unit, Veterans Administration, Regional Office, Portland. Foreword by Evarts A. Graham, M.D., Professor Emeritus of Surgery, Washington University, St. Louis. Cloth. Pp. 300, with illustrations. The Year Book Publishers, Inc., Chicago. 1954.

The practicability of planned intrathoracic surgical procedures has resulted in an increasing interest as well as an extensive literature in this important field. From this literature, the author has reviewed much of the new knowledge of the anatomy, embryology, congenital anomalies and surgical problems of the bronchovascular system.

The anatomical section contains an exhaustive, detailed analysis of the common bronchovascular variations not found in the standard text. Congenital heart disease is systematically discussed from the standpoint of the malformation and clinical findings but with emphasis on the surgical problem, solution and indications for surgical intervention. The experimental surgery of the pulmonary vessel, trachea and bronchi, the surgery of asthma and the technique of pulmonary resection is reviewed in the second half of the book. A series of line drawings illustrating unusual variations of pulmonary arteries and veins is to be found in the appendix.

The book is attractively bound and the paper and typography of excellent quality. For those interested in investigative work, the bibliography of over 500 references will serve as a helpful guide. Of interest primarily to the thoracic surgeon and bronchoesophagologist, this book should make instructive reading for the internist, radiologist and pediatrician as well.

P. H.

THE ASSOCIATION OF MILITARY SURGEONS OF THE UNITED STATES.

ANNOUNCEMENT.

The 61st Annual Convention of the Association of Military Surgeons of the United States will be held November 29 to December 1, 1954, at the Hotel Statler, Washington, D. C. The program will cover a wide range of professional and scientific subjects plus the latest advances in medical and allied sciences, the latter to be shown in a large variety of technical exhibits and films.

For the entertainment of the members and guests, a full schedule of events is being arranged. As in the past, one of the outstanding features is the Honors Night Dinner on December 1, at which the Sir Henry Wellcome medal and prize, the Gorgas medal, the Stitt award, the McLester award, the Louis Livingston Seaman prize, and the Founder's medal will be presented.

The granting of credit points for retirement to eligible Reserve Officers for attendance at meetings is expected to be announced soon by the Department of Defense.

THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC., MEETINGS.

MID-WINTER.

Eastern Section Meeting——————Friday, January 7, 1955
Philadelphia, Pa., Hotel Warwick

Triological Council Meeting——————Saturday, January 8, 1955
New York City, The Waldorf-Astoria

Western Section Meeting—Saturday-Sunday, Jan. 15-16, 1955
Los Angeles, Cal., The Town House

Southern Section Meeting——————Saturday, January 22, 1955
Charlottesville, Va., Medical School Auditorium

Middle Section Meeting——————Monday, January 24, 1955
Detroit, Mich., Sheraton-Cadillac Hotel

SPRING.

American Board of Otolaryngology
Richmond, Va., March 6-10, 1955

All Society Meetings will be held at the Hollywood Beach Hotel, Hollywood, Florida.

American Laryngological Association——————March 13, 14, 1955

Triological Society—(mornings only) March 15, 16, 17, 1955

American Broncho-Esophagological Association
(afternoons only) March 15, 16, 1955

American Otological Society, Inc.——————March 17, 18, 1955

The Hollywood Beach Hotel (American plan) is a delightful place to hold the Spring Meetings — you may wish to bring the family along. The Hotel offers its guests such features as ocean bathing, deep sea fishing, 18-hole golf course and country club, tennis and many other activities.

For hotel reservations please communicate with Mr. John W. Tyler, Manager, Hollywood Beach Hotel, Hollywood, Florida.

TWENTY-FOURTH ANNUAL MID-WINTER CLINICAL CONVENTION IN OPHTHALMOLOGY AND OTOLARYNGOLOGY, JANUARY 17 THROUGH JANUARY 28, 1955.

The Research Study Club of Los Angeles announces its Twenty-fourth Annual Mid-Winter Clinical Convention in Ophthalmology and Otolaryngology, January 17 through January 28, 1955. For the Ear-Nose-Throat, the main Guest Lecturers will include Dr. Theodore E. Walsh, Professor and Head of the Department of Otolaryngology, Washington University, St. Louis; and Dr. John J. Conley, Chief of the Head and Neck Service, St. Vincent's Hospital, New York City, and Clinical Professor of Otorhinolaryngology, New York University, and other appointments.

Dr. Walsh has long been known to us not only as a professor, but also as a lecturer in our national societies, with particular interest in otologic and rhinologic problems.

Dr. Conley has done a great deal of work in tumors and plastic surgery of the head and neck, as well as being a well trained man in the broader field of our specialty.

Also serving on our program with Didactic Lectures will be Dr. Kenneth C. Brandenburg of Long Beach, Dr. J. C. Almy Harding of San Diego, and Dr. Paul J. Moses of San Francisco.

Instruction Courses in Otolaryngology will be provided by Dr. E. R. V. Anderson, Dr. Norman Jesberg, Dr. Harold Owens, and Dr. Manuel R. Wexler, all of Los Angeles, and Dr. Leo Shahinian of San Francisco.

The main Guest Lecturers for the Eye program will be Dr. Edmund B. Spaeth and Dr. Irving H. Leopold, both from the University of Pennsylvania Graduate School of Ophthalmology.

The first week will be devoted to the Ear-Nose-Throat—from Monday to Saturday, January 17 through January 22. The Eye week will cover Monday to Friday, January 24 through January 28. Those of us who confine our work to only one of these specialties may complete either subject in one week.

On Tuesday evening, January 18, the Annual Banquet of the Los Angeles Society of Ophthalmology and Otolaryngology will honor the Guest Lecturers of that week. This will be held at 7:00 p.m. in the Ball Room of the Elks Club.

On Wednesday afternoon of each week motion pictures will be shown, Ear-Nose-Throat subjects the first week, and Eye subjects the second week. These afternoons will also afford time for relaxation, sight-seeing, golf, radio broadcasts, and the enjoyment of points of interest in and about Los Angeles, including the famous Huntington Library with its collection of rare books and art.

The American Laryngological, Rhinological and Otological Society, Western Section, will hold its Annual Meeting at The Town House immediately preceding this Convention, Saturday and Sunday, January 15 and 16. On Saturday afternoon the scientific session is open to members and all interested otolaryngologists. Saturday evening there will be a dinner meeting for members of the Triologic Society only. Sunday morning, the second scientific session is open to all interested. The president of the Society, Dr. Kenneth M. Day, after attending this Society's Annual Meeting, will be with us during a portion of the Mid-Winter Course of the Research Study Club.

Unless you have your own plans for a place to stay, it is advised that you write for reservations at an early date to Mr. H. M. Nickerson, Manager of the Elks Club, 607 South Parkview Street, Los Angeles 57. He will endeavor to arrange suitable quarters for you in a conveniently located hotel, and will confirm the reservation by return mail.

Each applicant must be a member, in good standing, of the American Medical Association *in Order to Become Eligible* for attendance at the Convention. The fee for the entire two weeks, or any part of it, is $100.00 and includes the cost of all luncheons. We are informed that these dues are an income-tax deductible item, as they represent an annual membership fee. Make your Check payable to "Mid-Winter Clinical Convention," and mail to Pierre Violé, M.D., Treasurer, 1930 Wilshire Boulevard, Los Angeles 57. If anything prevents your attendance, this fee will be returned to you.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

October 1, 1954.

Acousticon Models A-17, A-180 and A-185.

Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 1, New York.

Auditone Models 11 and 15.

Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67 and 70.

Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac Model M; Mono-Pac Model "Lyric"; Mono-Pac Model "Rhapsody."

Manufacturer: Beltone Hearing Aid Co., 2900 West 36th St., Chicago 32, Ill.

Cleartone Model 700.

Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Junior Model D-2; Dahlberg Model D-3 Tru-Sonic; Dahlberg Model D-4 Tru-Sonic.

Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Fortiphone Models 19-LR; 20A; 21-C and 22.

Manufacturer: Fortiphone Limited, Fortiphone House, 247 Regent St., London W. 1, England.
Distributor: Anton Heilman, 75 Madison Ave., New York 16, N. Y.

Gem Hearing Aid Model V-60.

Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.
Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico Model J; Maico Top Secret Model L; Maico Maxitone.

Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis, Minn.

Micronic Model "Mercury."

Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Model T-10; Microtone Model T-612.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

Normatone Model C and Model D-53.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.

Distributor: Normatone Hearing Aid Co., 22 East 7th St., St. Paul (1), Minn.

Otarion Models B-15 and B-30; Otarion Models F-1, and F-3; Otarion Model H-1; Custom "5."

Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40, Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny Myte).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Model 62 Starlet; Model 72; Model 82 (Zephyr).

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Distributor: Radioear Corp., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Silvertone Model H-16, J-92; Silvertone Model P-15.

Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.

Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966; Sonotone Model 977; Sonotone Model 988.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Televox Model E.

Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 99; Telex Model 200; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 953; Telex Model 1700.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.
Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster; Model Cameo.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Unex Models 200 and 230.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal; Zenith "Regent."
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices have vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TRANSISTOR HEARING AIDS ACCEPTED.

Acousticon Model A300; 1 transistor, 2 tubes. Model A-310; 1 transistor; 2 tubes. Model A-330; 3 transistors. Model A-335; 3 transistors.
Manufacturer. Dictograph Products, Inc., 95-25 149th St., Jamaica 35, New York.

Audivox, Model 71; 3 transistors.
Manufacturer: Audivox, Inc., 123 Worcester St., Boston 18, Mass.

Beltone Concerto Model; 3 transistors.
Manufacturer: Beltone Hearing Aid Co., 2900 W. 36th St., Chicago 32. Illinois.

Maico Transist-Ear, Model O; 3 transistors.
Manufacturer: The Maico Company, Inc., 21 N. 3rd St., Minneapolis 1, Minnesota.

Micronic "All American" Hearing Aid; 3 transistors.
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Model T1 (Red Dot); 3 transistors. Microtone Model T1 (Yellow Dot); 3 transistors. Microtone Model T31 (Micro-Mite); 1 transistor and 2 tubes. Microtone Model Skylark; 3 transistors.
Manufacturer: The Microtone Corporation, Ford Parkway on the Mississippi, St. Paul 1, Minn.

Otarion Model C-15; 1 transistor, 2 tubes. Otarion Model D-1; 3 transistors. Otarion Model F-22; 1 transistor, 2 tubes.

Manufacturer: Otarion, Inc., 4757 N. Ravenswood Ave., Chicago 40, Ill.

Paravox Model K.M. (TRANSonic); 3 transistors.

Manufacturer: Paravox, Inc., 2056 East 54th St., Cleveland, Ohio.

Radioear Model 820; 3 transistors.

Manufacturer: E. A. Myers & Sons, Inc., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Sonotone Model 1010; 1 transistor, 2 tubes. Model No. 1111; 3 transistors.

Manufacturer: Sonotone Corporation, Elmsford, N. Y.

Telex Model 954; 1 transistor, 2 tubes. Telex Model 956; 3 transistors.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Unex Model TR-3D; 3 transistors.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Zenith Model Royal-T; 3 transistors. Zenith Model Super Royal-T; 3 transistors.

Manufacturer: Zenith Radio Corp., 6801 W. Dickens Ave., Chicago 39, Illinois.

SEMI PORTABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).

Manufacturer: A. M. Brooks Co., 1222 W. Washington Blvd., Los Angeles 7, Calif.

Aurex Hearing Aids (three types).

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.

Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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REVIEW OF AVAILABLE LITERATURE ON THE PHARYNX AND PHARYNGEAL SURGERY FOR 1953.*

FRANC[illegible] E. LEJEUNE, M.D.,

[illegible] LEWIS, JR., M.D.,

and

FRAN[illegible] NORMANN, M.D.

New Orleans, La.

ANATOMY AND PHYSIOLOGY.

Bosma states that interest in the motor functions of the pharynx has recently been stimulated by the increasing incidence of bulbar-pharyngeal poliomyelitis. The accumulation of patients having residual difficulties of deglutition and speech, sequelae of this form of poliomyelitis, impelled Bosma to study further the motor mechanisms of the pharynx and to seek new methods of correction of these particular handicaps. In an excellent, well illustrated article, Bosma shows the methods used in the study of cadaveric material as well as the anatomic observations made on living patients whose pharynges had previously been exposed by surgical resection of neoplastic or osteomyelitic tissue in the area of the nose and paranasal sinuses. The anatomy and function of the musculature of the upper pharynx have been carefully studied. The position and action of the levator veli palatini, pharyngopalatinus, salpingopharyngeus and superior constrictor muscles and a number of inter-relations of their action have been identified. The commonly observed action of the pharyngopalatinus, salpingopharyngeus and superior constrictor muscles was their combined action of elevation and constriction of the upper pharynx. The levator veli palatini muscle was identified as a prime mover in this combination of action of the intrinsic muscle of the pharynx.

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* From the Department of Otolaryngology, [illegible], New Orleans.

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Palatopharyngeal closure is accomplished by the tensor veli palatine, levator veli palatine, superior constrictor of the pharynx, salpingopharyngeus and pharyngopalatinus. The main function of this muscle valve is occlusion or constriction of the passageway between the oropharynx and the nasopharynx during speech and deglutition. Bloomer[2] has made an extensive study of the movements of these muscles during speech, swallowing, and other mechanisms in two patients with surgical facial defects. Sound and silent movies were made. An excellent record of these observations is presented, which should be of value to the oral surgeon, the prosthodontist and speech pathologist.

Vorhaus and Deyrup[3] describe the accelerating action of ATP on the ciliary movement of the frog's pharyngeal mucosa as "highly consistent, rapid, reversible and demonstrable in the presence of relatively low concentration of ATP." It seems to have a specific effect in that "hydrolysis of the substance abolishes completely its effect on the test system." The mechanism of action of ATP on ciliary movement remains to be clarified and, the authors believe, deserves "careful reinvestigation in the light of recent developments in the field of cell physiology and biochemistry."

PATHOLOGY.

Semenov[4] gives an excellent discussion of the pathology of the pharyngeal recess, Eustachian tubes, tonsils and adenoids from a clinical viewpoint. The pathology of lymphoid tissue is described in detail. The importance of the nasopharyngeal pituitary gland is stressed. The advantages of careful surgical procedures on the adenoid combined with nasopharyngeal radium application over indiscriminate Roentgen-ray therapy to the nasopharynx is stressed. This is an excellent article and should be read in its entirety by all otolaryngologists.

ANOMALIES.

In delivering the Hunterian lecture, Hynes[5] stated that after cleft palate operations speech can be excellent; however, sometimes unsatisfactory results are obtained, usually in patients with an enlarged nasopharynx and a palatopharyngeal space which does not close properly. Hynes, therefore, devised a pharyngoplasty by muscle transplantation to correct the abnormality in these cases. This technique is described and illustrated.

The physiology of the pharynx in relation to voice production is discussed and evaluated. He concluded that in cleft palate in which the result of operation is considered a failure, if the soft palate remnants are reasonable in size and mobility, it is possible to produce a palatopharyngeal sphincter and excellent speech can be expected. If the soft palate remnants are hopelessly small, pharyngoplasty by muscle transplantation should be done.

In a survey of the American literature, McGovern[6] could find no record of the association of congenital choanal atresia with congenital heart disease. In a previous paper McGovern had discussed the physiologic and hereditary aspects of choanal atresia and mentioned associated abnormalities, such as high arching of the palate, asymmetry of the face, bifid uvula, congenital coloboma of the iris, double tragus, double congenital aural fistula, polydactylism and flat nose. In the present article he adds to this list two cases of choanal atresia associated with congenital heart disease, which are presented in detail. As evidence that this congenital anomaly has a hereditary tendency, McGovern cites a case reported by Dixon, of bilateral choanal atresia in twin sisters.

A pharyngeal flap taken from the posterior pharyngeal wall and attached to the posterior surface of the vellum is one of the methods used to lengthen the palate to improve speech. The problem of anesthesia in this limited surgical area is great. Walden and associates[7] have used preliminary tracheotomy and intratracheal anesthesia in eight patients with success.

DIVERTICULA.

Perzik[8] tells us that since 1884 when the surgical treatment of pharyngeal pulsion diverticula was first employed, the vertical incision has been the avenue of approach. This incision has usually extended from the level of the hyoid bone to the suprasternal notch along the anterior border of the sternomastoid muscle. Perzik is of the opinion that such a vertical incision is a handicap and is limited in the transverse diameter by limited retraction. For this reason he advocates the usual thyroid collar incision, which begins 2 cm. above the clavicle and is carried in a curved line 3 cm. beyond the median line on the right side and 3 cm. beyond the posterior border of the sternomastoid muscle. Illustrations are utilized to demonstrate the new approach, which results in better exposure because of the improvement in the transverse diameter of the field. The reviewers also believe that the collar incision produces a superior cosmetic effect to that of the vertical incision in the neck.

The late Dr. Lahey[9] had the largest published series of surgically treated pharyngoesophageal diverticulas. His mortality rate was the lowest of any large series, consisting in two deaths in 359 patients. Lahey attributed his success in the management of these patients primarily to the use of a two-stage procedure. He gives a lucid description of the surgical technique he employed.

Perzik[10] gives a brief discussion of pharyngoesophageal pulsion diverticula. He describes his one-stage technique for excising the diverticulum using a transverse collar incision.

Phelps[11] presents a discussion of 24 cases of pharyngeal diverticula, 20 of which were treated surgically with no deaths. Serious complications developed in 12 patients. These included fistula, stricture at the site of excision, recurrence, thoracic complications and unilateral vocal cord paralysis. There were no cases of infection. Phelps advises surgical removal of the sac as soon as the diagnosis is made, as the smaller the sac, the easier the performance of the operation. He believes that complications can be prevented by "earlier diagnosis and operation while the sac is small; by better preoperative care; by better surgical technique, including gentle manipulations of the strictures; by putting less pull on the laryngeal nerves; and by accurately suturing the mucosa and muscle layers." He recommends elimination of the feeding tube and intravenous feeding for the first few days.

DISEASES.

Denny and coworkers[12] compared the effects of penicillin, aureomycin and terramycin on acute streptococcal pharyngitis and tonsillitis in a well controlled series, without the benefit of sensitivity determinations. These drugs shortened the clinical course somewhat over the control cases. Leucocytosis and antibody formation were lowered. Undesirable side effects were more common in the patients treated with aureomycin and terramycin.

Dintenfass[13] reviews in an orderly fashion the basic concepts of etiology and treatment of chronic pharyngeal infection and postnasal drip. He gives his ideas regarding treatment of the various syndromes, which for the most part, conform with generally accepted views. The importance of ventilation and drainage, plus allergic management is stressed. This article should be of value to basic students in otolaryngology.

Loeb[14] presents his experience with a series of 100 patients with epipharyngeal crypts. They are more common than presently thought and should be looked for in any patient complaining of sore throat, lump in the throat, headache, postnasal drip and crusts, blood in the throat and cough. These crypts appear as a rounded, smooth-bordered, clear-cut hole in the midline of the epipharynx, just above the level of the palate. Relief from symptoms should follow anesthetization of the crypt. In most of Loeb's cases, tonsillectomy had already been done in an effort to relieve symptoms. Treatment is directed toward obliteration or saucerization of the crypt.

Tyler[15] reports the case of a 16-year-old white girl who complained of a clicking and cracking noise in the head, migratory at first but eventually localized in the posterior portion of the mouth. It was accompanied by a grating noise in the top of the head. Physical and neurologic examinations yielded essentially negative results except for the palatal movement. With the mouth open a rhythmical 72-minute movement of the palate and uvula in a superior and inferior direction was found to be constantly present with audible clicking. The patient had no difficulty swallowing, no weakness and no paralysis of facial muscles. Fluoroscopy revealed normal diaphragmatic movement.

This is considered a typical example of myoclonus due to damage in the central tegmental tract affecting the primitive gillslit respiratory muscles. The myoclonus resembles the gill movements of fishes. It does not extend into the somatic musculature and is likely to clear up spontaneously in a young individual.

Zinneman and Hall[16] report an interesting case of pharyngeal and laryngeal infection with Histoplasma capsulatum. A 42-year-old man noted the gradual onset of hoarseness and dyspnea necessitating tracheotomy. Because of severe sore throat deglutition was painful, resulting in loss of 25 pounds in weight. There was generalized swelling and reddening of the mucosa of the hard palate, tonsillar pillars, uvula and posterior pharyngeal wall. The mucosa was granular in appearance and of spongy consistency. There was no ulceration. Similar lesions were noted in the larynx. Biopsies revealed the characteristic histologic picture of histoplasmosis. Ethyl-vanillate 0.6 Gm. in capsules was given orally every three hours. In six weeks there was no evidence of histoplasmosis.

The natural reservoirs of Histoplasma capsulatum are the soil, dogs, rats, skunks, opossums and cats. Until recently, human histoplasmosis was thought to be a fatal infection. Clinical manifestations vary. Ethyl-vanillate is a comparatively new drug, having been used only for the past two years, and apparently successfully copes with stubborn infections of histoplasmosis. The drug is not without danger and should be used carefully.

A case of histoplasmosis involving one vocal cord was seen by one of the reviewers in 1947. This patient was successfully treated with promine and remains well today except that Addison's disease has developed as a complication of the histoplasmosis. We should be grateful to Zinneman and Hall for calling attention to the use of Ethyl-vanillate in infections of histoplasmosis.

Bucco-pharyngeal ulcerations of undetermined etiology form the basis of an interesting discussion by Harrison[17] These recurrent ulcerations are not only painful but they also frequently defy all manner of treatment. The lesion may appear anywhere on the mucous membrane of the buccal cavity and pharynx, usually as a small red papule about the size of a pin head. Ulceration with a yellowish exudate forms rapidly and pain develops with equal rapidity. These ulcerations may be single or multiple and occasionally are fairly large. Harrison was able to obtain no information as to the etiologic factor from all laboratory tests including scrapings and biopsies. Most of Harrison's patients gave a history of severe emotional upset preceding the onset of the lesions. Numerous therapeutic agents were employed, but best results were obtained from local application of a paste made from a 0.25 mg. capsule of aureomycin in glycerine. Pain was relieved in 24 hours. The use of small doses of Roentgen-ray was of questionable value. Fortunately, such cases are relatively rare. Failure to establish the etiology of the ulceration, particularly when large, is at times disconcerting. In severe cases, or in extensive ulcerations defying all methods of therapy we have successfully used cobra venom, when available, or smallpox vaccine.

Meyrick[18] reports the case of a patient with symptoms typical of the syndrome of the sinus of Morgagni, the physical signs of which were originally described by Trotter in 1911. The patient had lost hearing in the left ear for the past two-and-one-half years and complained of vague nasal symptoms for a period of time. In the past two years he had had pain on the left side of the face, maxilla and ramus of the left mandible, and for four months, diplopia and difficulty in moving the jaw. A tumor was present in the nasopharynx and a biopsy taken through the nasal passage established a diagnosis of malignant endothelioma. Deep Roentgen-ray therapy was instituted, and 12 months later all signs and symptoms had subsided except a slight degree of deafness in the left ear.

Delaney and Nelson[19] consider the anginose, or pharyngeal type, to be the most common of the many cases of infectious mononucleosis seen. Infectious mononucleosis is frequently confused with acute tonsillitis, peritonsillar abscess, acute pharyngitis, Vincent's infection, or even diphtheria. The anginose type of infectious mononucleosis, according to Delaney and Nelson, is characterized by diffuse pharyngitis and tonsillitis.

Prostration is no greater than in simple tonsillitis or pharyngitis, and severe respiratory difficulty is uncommon. Delaney and Nelson report a case of infectious mononucleosis which required tracheotomy. Decannulation was possible on the fourth day because of good response to terramycin, cortisone and gamma globulin. Cortisone was given for the possible effect on edematous pharyngeal tissue, but no reason is given for the administration of gamma globulin.

Wolf[20] correctly observes that prior to the advent of antibiotics, pharyngitis was treated primarily by warm gargles or application of a solution to the posterior pharyngeal wall. Instead of eliminating these methods the antibiotics have complicated the treatment of this infection. Troches are now available containing almost any desired antibiotic or groups of antibiotics with a local anesthetic. Since sensitization frequently arises from prolonged frequent contact with an antibiotic, the local use of any systemically employed antibiotic is contraindicated. Reaction to the "caine" group of anesthetic agents is not uncommon. Such reactions may take many forms, and the symptoms of pharyngitis may persist as a result of the use of these drugs. Wolf concludes that since parenteral medications are so often used successfully in infections, it is probably best to avoid intraoral use of any possible sensitizing agent, whenever possible.

In a preliminary survey of persistent sequelae of bulbar poliomyelitis an unexpectedly high incidence of persistent or recurrent disability of the pharynx was noted by Bosma.[21] In an excellent presentation he further states that two patterns of partial paralysis and associated disability of function have been observed. The more common is that of weakness of the levator veli palatini muscle with associated impairment of the palatopharyngeal sphincter. This results in a nasal quality of speech and possibly in nasal regurgitation of fluids. The less common but more critical paralysis is that of the pharyngeal constrictor musculature associated with inability to swallow. These problems are further discussed in a highly interesting and scientific manner. The material presented by Bosma is too lengthy to review here, but the article should be read by all interested in the subject, as Bosma has devoted much time, thought, and study to this important problem.

Hansel[22] presents five cases of patients with somewhat vague symptoms referred to the arterial region of the pharynx and larynx as convincing evidence that these phenomena are vascular in nature. The clinical picture resembles that of migraine, tension and histaminic headaches. Hansel believes that the response to the administration of histamine and the symptomatic relief from the use of ergot preparations indicate that in certain patients pharyngeal and laryngeal symptoms may be of vascular origin.

Wallach and associates[23] describe an extremely contagious, previously unreported disease of children. The clinical course follows a typical pattern. The incubation period is 24 to 72 hours. During the first 36 hours the only clinical finding is a scattering of small vesicles or petechiae on the hard and soft palate and frequently on the pharynx. Following this, however, high fever, cervical lymphadenopathy and grippal symptoms rapidly develop. During this stage, coalescence of the smaller petechiae may produce large hemorrhagic blotches. The temperature usually terminates by crisis in four or five days, the mouth remaining sore for three or four more days. Penicillin therapy has no effect whatsoever. Terramycin, if administered prior to the onset of fever, seems to reduce the severity of the ultimate clinical course; however, after the onset of fever, it apparently has no effect. No laboratory studies were made during this epidemic. Hospitalization was not required; there were no fatalities and no serious complications.

Recent studies have indicated interference with the action of penicillin or streptococci by chloramphenicol. Walker[24] was unable to demonstrate this interference in a controlled series of 71 patients with acute streptococcal pharyngitis. The series was divided into three groups: one received penicillin in usual doses, and chloramphenicol in maximum doses; the second, penicillin, and chloramphenicol in minimal doses; the third, penicillin alone.

Waldo[25] states that the most common cause of acute pharyngitis is non-bacterial, the so-called "viral" pharyngitis, and requires only symptomatic treatment. Diphtheritic sore throat, which is still encountered in Utah, is preventable by immunization; early diagnosis and administration of antitoxin are imperative. Acute streptococcal sore throat presents a beefy red, edematous pharynx and responds readily to penicillin. The acute pharyngitis seen in infectious mononucleosis is commonly undiagnosed. A high index of suspicion and a positive heterophil agglutination reaction aid in diagnosis. Vincent's pharyngitis presents dirty, punched out, ragged ulceration and responds to administration of penicillin. Secondary syphilis may produce acute pharyngitis. Diagnosis in such cases is important, because insufficient penicillin therapy is harmful to the course of the disease.

Kinghorn and associates,[26] an internist, a laryngologist and a bacteriologist, report good results in 16 patients with chronic sinus disease and seven patients with "nasal catarrh" treated with autogenous vaccine and filtrate, both intradermally and locally. This study apparently took place over a period of many years.

Asherson[27] describes a characteristic syndrome manifested by a characteristic pharyngogram. This syndrome is produced by incoordination or failure of relaxation of the cricopharyngeus muscle (achalasia) during the act of swallowing. Any lesion that interrupts the persistaltic wave preceding a swallowed bolus will produce this syndrome. Paralysis of the pharyngeal plexus following poliomyelitis, paralysis of one or both recurrent laryngeal nerves, and pharyngotomy are common causes. There may be no symptoms, or symptoms may be so severe that gastrostomy is required. This is strictly a roentgenologic diagnosis. Differential diagnosis includes postcricoid carcinoma and pharyngoesophageal diverticulum. "Globus hystericus" is not mentioned. Numerous case histories and Roentgenograms are included.

According to Cockburn,[28] during August and September, 1951, 205 cases of acute nonpurulent conjunctivitis, vesicular pharyngitis, muscular ache and fever appeared in Greeley, *Colorado. Cockburn named this syndrome "Greeley's disease."* The condition affected children and teen-agers and seemed to be transmitted by contact or by association with a swimming pool. No etiologic agent was found. Similar epidemics occurred during the same months in Missouri and other areas of Colorado. Many of these patients were suspected of having poliomyelitis, but the spinal fluid examinations yielded normal results. There were no complications of the disease.

Kinawi[29] reports two interesting cases of primary atypical pneumonia in sisters in whom a peculiar painless, afebrile, sterile membranous lesion of the tonsils developed ten days after resolution of the pneumonia. Both cases were thoroughly studied etiologically and clinically, and no causative agent could be found. Kinawi suggests the term "primary atypical pulmonary tonsillar syndrome" for this condition.

BENIGN TUMORS.

Davis[30] reports a case of benign pharyngeal polyp in a 19-year-old girl which caused her to complain of a feeling of "something in her throat," gagging, vomiting and loss of weight of two years' duration. Examination revealed a large, white glistening mass extending behind the tongue into the hypopharynx. This was removed by a snare. It measured 7.5 x 3 x 1.5 cm. and was attached by a narrow pedicle to the left lateral wall of the nasopharynx. There has been no recurrence.

Guggenheim[31] *reports a case of schwannoma of the pharynx,* which is considered a rare disease. He was able to find only 34 similar cases reported in the domestic and foreign literature and these, which are tabulated, form part of this excellent paper. In the case reported the tumor extended from above the soft palate to just below the level of the epiglottis and after preliminary tracheotomy a submandibular incision on the left was made back to the angle of the jaw curving gently around to continue downward along the anterior border of the sternocleidomastoid muscle. The external carotid was ligated and dissection continued until the tumor was exposed and removed. Upon intraoral examination later, Guggenheim discovered some of the tumor still present, and this was removed perorally. The origin of the tumor is discussed, and Guggenheim advocates a combined approach in all cases of schwannoma of the pharynx.

According to Tingwald[32] only 20 cases of polyps arising in the oropharynx and hypopharynx have been recorded in the literature. He describes an additional case of a 48-year-old white man with a one year history of a lump in the left side of his throat which caused him to have a constant desire to clear his throat. The lump was caused by a polyp whose pedicle was attached to the left lateral pharyngeal wall at the level of the arytenoid cartilage. After fixation the specimen measured 4.5 cm. in length and 1.5 cm. in diameter. Previous pathologic diagnoses of these lesions include lipoma, myxoma, fibroma, fibro-epithelial polyp and benign fibromatous polyp. In two of the reported cases the lesions were multiple. Symptoms reported include regurgitation of the polyps, intermittent dysphagia, lump in the throat, throat clearing, cough, dyspnea, hoarseness, wheezing, nausea and vomiting. Three patients died of asphyxia due to aspiration of these polyps and it is primarily for this reason that they achieve importance.

Farrar[33] classifies fibromas of the nasopharynx into two distinct types: the first is the simple fibroma, which may arise in the nasopharynx or grow into it from the nose. It has a variable histologic picture, is vascular, may occur at any age and in either sex. The second type is the juvenile basal fibroma of the nasopharynx. It arises only in the nasopharynx and occurs nearly always in adolescent boys. The site of attachment is usually on the sphenoid. These tumors are very vascular, the blood vessels lacking muscular wall in parts. Occasional surgical removal may be accompanied by uncontrollable hemorrhage.

Farrar reports a case of simple fibroma of the nasopharynx occurring in a boy aged 13 years. The tumor, which was smooth and firm to touch, filled the nasopharynx and was attached to the posterior tip of the right inferior turbinate. It was readily removed by snare and a brisk hemorrhage was controlled with packing. Such tumors are comparatively rare, only six being found in a review of 211 tumors of the nose and *nasopharynx.*

In an interesting article on benign neoplasms of the nose, Handousa[14] presents the *main* features in 73 consecutive cases seen in private hospitals. The tumors encountered were osteomas, hemangiomas, fibromas, papillomas, chondromas, lymphomas and myxomas. Each of these groups of cases is decribed accurately, and the operative procedure and results are tabulated. Comments pertinent to the type of tumor described are offered.

A new surgical approach to *fibromas* of the nasopharynx [illegible] described and illustrated by Kremen [illegible] scribing his operative approach, he [illegible] giofibromas or the so-called juvenile [illegible] rynx and correctly states that they occur [illegible] young pubescent males. The usual symptoms produced by these tumors are nasal obstruction [illegible] ing, and frequent *bleeding* [illegible] lesion grows rapidly during adolescence, [illegible] the prominent *vascular channels tend to become* smaller, the tumor regresses considerably. Although the lesion is not malignant histologically, its growth and *expansion* are at times responsible for changes *in facial contour. It is well known* that these tumors usually arise from the posterior superior wall of the nasopharynx. *Because they do not respond* satisfactorily to radiation and continue *bleeding* excessively, it frequently becomes necessary *to remove them surgically.* Because Kremer was critical of operations *heretofore* used for removal of nasopharyngeal fibromas, he devised a technique which is described and *illustrated.* He *makes* a vertical incision in front of the ear and *down the neck just* anterior to the sternomastoid muscle. Another incision beneath the angle of the mandible is of assistance in *retracting flaps* and exposure. The external *carotid* is ligated. The masseter muscle is exposed and incised to the *periosteum,* which is elevated, and the mandible is then transected with a Gigli *saw about* 1 cm. below the notch formed by the coronoid and condyloid processes. The edges of the cut mandible and the pterygoid muscle fibers are separated. This exposes the [illegible] wall of the nasopharynx, which is incised longitudinally on its lateral wall. The tumor is thus exposed and extirpated. The mandible is brought together with stainless steel wire. Two cases in which this operation was successfully employed are reported.

Much credit is due Kremen for the development of a new technique which he claims is advantageous; however, the technique appears complicated and long in contrast to the time honored methods which have served excellently in the past. We have had opportunity to see many juvenile nasopharyngeal fibromas, and all presented problems. Controlling hemorrhage has been the most important, and when accomplished, we have found that repeated electrocoagulation and repeated implantation of radium needles or radon seeds into the tumor have eventually reduced the vascularity and size of the tumor so that it could be successfully removed by snare.

An unusual case of transitional cell papilloma occurring on the nasal and oral portions of the post-pharyngeal wall is reported by Radcliff.[36] This tumor, which extended from the basisphenoid to about one-inch above the epiglottis, appeared as a papilliferous mass attached to the posterior pharyngeal wall. Through a transverse palatine incision the growth was removed by diathermy excision without difficulty or complications. The pathologic report was transitional cell papilloma with no evidence of malignancy. Radcliff reviews the literature on similar cases showing that there is some definite difference of opinion on the histologic aspects of this type of neoplasm.

Manning[37] reports the case of a 25-year-old man with a solitary neurofibroma bulging into the pharynx and neck associated with Horner's syndrome. The mass was removed, and there was no evidence of recurrence eight months later. Five other cases collected from the literature are reviewed.

MALIGNANT TUMORS.

Dysphagia associated with hypochromic anemia, which has been called Plummer-Vinson syndrome, Patterson-Brown-Kelly syndrome, and sideropenic dysphagia, is common in Sweden.

Lindvall[38] gives an excellent discussion of the etiology, pathology and typical roentgenographic observations in such patients. In 300 cases of hypopharyngeal or upper esophageal carcinoma observed in women, over 90 per cent gave a history of dysphagia and *hypochromic anemia*. Valve-like obstructions, webs and actual strictures are demonstrated in the hypopharynx and upper esophagus roentgenologically in many patients. When a carcinoma occurs, it is practically always proximal to these changes. Eight cases are presented in which roentgenologic changes of sideropenic dysphagia were demonstrated many years prior to the development of the carcinoma.

In a review of the literature Holinger and Rabbett[39, 40] found nine cases of malignancy of the pharynx and larynx in which the tumor developed in an area previously irradiated for a benign process and present three cases of their own. The latent period appears to be 20-35 years. Cases of carcinoma of the skin developing in previously irradiated areas are well known. Recently, reports of bone sarcomas in areas treated three to 12 years previously for tuberculous arthritis and carcinoma of the cervix developing ten or more years after irradiation therapy for a benign lesion of the cervix have appeared in the literature. Since irradiation therapy was more common for benign conditions of the neck several decades ago, more of these cases may be seen in the immediate future. They believe the sex distribution of the 12 cases may be significant (nine females and three males), since the pyriform sinus and the laryngeal lesions are seen most commonly males and only the postcricoid lesions most frequentl females.

Vincent[41] reports the case of a patient aged six years, who died of a rapidly growing tumor of the nasopharynx two months following tonsillectomy. An extensive neuroblastoma arising in the nasopharynx, filling the upper air and food passages, and extending into the cranial cavity at several points was found at autopsy.

Rubenfeld and Winston[42] state that obtaining cytologic cells by suction has found its greatest application in [illegible] tumors. The simplicity of the procedure prompted them to try the same technique in tumors of the nasopharynx. With the patient in the sitting position the nasal cavity is cocainized and a metal cannula is inserted along the floor of the nose until it strikes or comes in contact with mucosa or tumor. Suction is produced by a 55 cc. syringe as the cannula is moved around, and the trauma produced by the pressure and motion of the cannula is sufficient to obtain a few fragments of tissue. The simplicity of the technique recommends its use.

In an excellent article Hultberg[44] describes Radiumhemmet's method of treatment in hypopharyngeal cancer. He points out the importance of first distinguishing between cancer of the upper and lower portions of the hypopharynx, the line of division being at the level of the upper border of the cricoid cartilage. The upper group comprises carcinoma of the edge and anterior surface of the epiglottis, the aryepiglottic fold, the arytenoid region and the pyriform fossa. To the lower group are assigned postcricoid carcinomas and tumors occurring at the entrance of the esophagus. Since 1930 fractionated but not protracted Roentgen-ray treatment has been used at Radiumhemmet. The technique is described in detail, one field being radiated each day, and it is estimated that the total dose required to produce complete regression of the cancerous growth is from 5700 to 6000 R. administered over a period of 28 to 32 days. Between 1939 and 1947, 322 (119 males, 203 females) patients with hypopharyngeal cancer were treated in this way. The incidence of five-year cures in the entire series was between nine and 10 per cent; however, in the 65 patients treated in 1948 and 1949, the five-year cure rate increased to 14.5 per cent. This indicates that the results are improving. Carcinomas in the lower part of the hypopharynx are usually regarded as incurable. Hultberg concludes that if it were possible to persuade people to desist from smoking and drinking strong spirits, fewer cases of pharyngeal carcinomas would be seen.

Welin[45,46] informs us that in Sweden the hypopharynx is a relatively common site for cancer. Since 1940, 510 such cases were seen at the Karolinska Hospital. Their records show that the malignancy in men is more often localized in the upper portion of the hypopharynx whereas in women the opposite is true. This is significant and probably connected with the relatively high incidence of the Plummer-Vinson syndrome in women, which is considered by Welin to be a precancerous condition. Tumors localized in the postcricoid portion of the hypopharynx are best diagnosed and their limits defined by radiologic examination. Since radiologic techniques differ in the various countries, Welin describes the technique used in Sweden. This is illustrated with diagrams and radiologic views. The diagnosis of carcinoma of the hypopharynx is made radiologically, and the extent of the tumor and the degree of invasion into the normal surrounding tissues is indicated to the clinician. Patients with Plummer-Vinson syndrome are requested to return every six months for follow-up examinations. This has resulted in the diagnosis of carcinoma before any new symptoms are produced. Roentgen-ray therapy is discussed, and a warning regarding late reactions following irradiation is sounded.

Duncan[47] reports a case of mixed tumor of the hypopharynx treated by local excision. Local recurrence, spread and pulmonary metastasis developed subsequently, and were treated palliatively with Roentgen-ray therapy.

Heatly[48] reports two cases of plasma cell tumors involving the maxillary sinus, both of which were remarkably vascular.

Plasma cell tumors involving the upper air passages are rare and are commonly associated with multiple myeloma. They may occur as localized submucosal tumefactions or as pedunculated polyps. They are quite unpredictable in behavior, and neither gross nor microscopic appearance is of value in predicting the degree of malignancy. The consensus is that they are best treated by Roentgen-ray therapy and local excision when this does not involve a radical surgical procedure. They should be considered malignant. Local and distant recurrences are possible many years after apparent cure of the initial lesion. All patients with plasma cell tumor should have a skeletal survey, marrow studies and a search for Bence-Jones protein to differentiate it from multiple myeloma.

Negus[49] believes that for carcinoma at or near the mouth of the esophagus, involving the posterior surface of the larynx, radical removal of a segment of the pharynx and upper end of the esophagus, together with the whole of the larynx is the only adequate surgical measure.

The primary principles involved include removal of the growth in one block as soon as possible. No preliminary measures to reconstruction are taken before the carcinoma has been eradicated. The entire procedure including the reconstruction is done in one stage.

The *primary* reconstruction is accomplished by filling the food passage defect with a plastic tube, tapered from above downwards, covered with a free split-skin graft. This plastic stent is allowed to remain for two months.

Complications include infection, fistula and stenosis. Biotherapy is largely capable of preventing infection. Stenosis may be benefited by bouginage but, preferably, the lumen should be re-established by formation of flaps.

Of 13 patients seen since 1949, only seven were considered potentially curable. Of these, two died one year after operation from local recurrence, and the other five are well three and one-half, two and one-half, and two and one-quarter years after operation.

Moore and Faulkner[50] call attention to the fact that extrinsic carcinoma of the larynx is amenable to pharyngolaryngectomy in only a limited number of cases and then rarely with a permanently successful result. Patients should be selected carefully with the idea of accomplishing both surgical extirpation of the lesion and plastic repair of the esophagus and pharynx in the shortest possible time.

The advantages and disadvantages of the Negus method of single-stage repair are discussed. It appears that the disadvantages could be overcome merely by improving existing techniques. In patients who survive the disease and the operation, stricture of the newly formed food passage may become a problem whose solution lies in several choices of surgical techniques. Four such methods are discussed in some detail. Moore and Faulkner describe their method of two-stage reconstruction in which the skin flaps to be used in the repair are sutured to the esophagus before it is cut across. In this way the cut end of the esophagus is "fixed" and does not retract, making anastomosis as low as the level of the aortic arch possible. The second stage is done as soon as the wound has healed and the sutures are removed. This method of reconstruction is particularly suitable in those patients whose tracheal defect is a major problem.

Irradiated skin should never be used in the repair. Fistulas, if small, will usually close spontaneously if the passage of food is diverted via a Levine tube. Larger fistulas may be closed by turning in local flaps. It is important to look for the development of hematomas beneath the skin flaps postoperatively. All saliva should be ejected into a sputum bowl and not swallowed.

Anesthetic problems and difficulties associated with this procedure are discussed.

Cade,[11] who has treated 403 cases of carcinoma of the pharynx in the past 22 years, believes that Roentgen-ray therapy offers little in its treatment. He considers radical surgical excision including the larynx, upper esophagus and pharynx the treatment of choice in operable cases. Raven discusses his technique of radical two-stage operation used in 24 patients. Three methods of closure of the pharyngoesophageal defect are available: partial closure with the original skin flap, delayed closure by tube pedicle grafts, or immediate closure by the use of a skin graft over a plastic tube. The latter is the ideal.

Patients with advanced cancer of the oropharynx, hypopharynx, larynx and cervical esophagus have a miserable terminal illness and death. Palliative surgical procedures with or without irradiation rarely improve their lot. Braund and Cocke[12] believe that in selected cases extensive surgical excision, with *en bloc* removal of the larynx, upper esophagus, portions of the oropharynx and hypopharynx as well as unilateral or bilateral radical neck dissections, not only provides satisfactory palliation but also offers hope for cure. Reconstruction of the upper esophagus, re-establishing pharyngoesophageal continuity, is accomplished by the use of parallel tube grafts raised from the anterior thoracic wall. Details of technique, and preoperative and postoperative care are given.

Gooddy[53] emphasizes the importance of careful inspection of the nasopharynx in cases of obscure facial pain. The initial symptoms of nasopharyngeal cancer are neurologic in 16 per cent of cases, Vth Nerve symptoms being by far the most common. Cawthorne[54] reviews the diagnostic procedure employed in tumors of this area. Professor McWhirter[55] stated that cancer of the nasopharynx is a rare lesion and not frequently seen by the medical students or by general practitioners.

The pharyngeal grooves are the terminal portions of the lateral food channels for directing food past the larynx into the esophagus. In an interesting article on cancer of this area, Lederman[56] points out the difficulties of trying to define the pharyngolaryngeal groove and particularly the sinus pyriformis for purposes of classifying tumors in this region. He concluded that the acceptable anatomic boundaries for the pharyngolaryngeal groove extend from the pharyngoepiglottic fold to the esophageal opening at the level of the lower border of the cricoid cartilage. He further attempted to distinguish tumors arising in the pharyngolaryngeal groove or "pyriform fossa" into an upper group and a "worse" or lower group. Signs and symptoms occurring in each group are briefly discussed as well as the radiologic appearances. During the years 1933-1951, 318 laryngopharyngeal tumors were seen and of these 119 or 37 per cent arose from the sinus pyriformis. Lederman states that teleradium has been the treatment of choice, only three patients in his series having undergone laryngopharyngectomy after radiation. The incidence of three-year survivals was surprisingly good, but it diminished considerably for survival from five years, owing to recurrences or metastasis. Only 6.5 per cent survived five years. In review, it must be remembered that cancer of the pyriform fossa, when first seen by a laryngologist is as a rule extensive, as the signs and symptoms are such that early diagnosis is very unlikely. Many of the patients treated by radiation have such extensive lesions that they are inoperable and, therefore, any survivals represent good salvage.

According to Conley[37] a one-stage operation for the treatment of patients with extensive cancer of the pharynx, hypopharynx, postcricoid areas and cervical esophagus has been developed. It consists in immediate re-establishment of continuity between the fenestrae in the oral pharynx and cervical esophagus by anastomosing a free, thick-split, tubed skin graft to these structures.

Indications for this procedure are extensive cancer of the cervical esophagus, of the pharynx and hypopharynx, and of the extrinsic larynx and pharynx. Plastic reconstruction is indicated whenever the cancerous process occupies more than 70 per cent of the circumference of the pharyngeal food passage. In esophageal lesions it is rarely possible to do less than total circumferential excision. The operation is not warranted in areas that have been heavily irradiated or are grossly secondarily infected.

Important steps in the technique include careful end-to-end anastomosis of the free skin tube to the circumference of the pharyngeal and esophageal mucosa. The longitudinal suture line of the tube is approximated to the prevertebral fascia of the unoperated side of the neck. This insures maximum support and nourishment to the tube which is also enhanced by the covering inferiorly with the lobes of the thyroid.

Complications include infection, fistula and stenosis. The latter is the most serious and may be prevented largely by use of plastic or wire mesh stents as an onlay for the tubed skin graft.

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THE MANAGEMENT OF EPISTAXIS.

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Epistaxis is not a new and interesting condition, and there is nothing startlingly new and interesting to report about its management. It is such a common condition, and fortunately, in the usual instances so simple to deal with, that we sometimes tend to forget that occasionally bleeding from the nose is of serious importance and may require the best medical and surgical skill and acumen for solution of the problem it presents. In rare cases when the hemorrhage is profuse or prolonged, epistaxis may constitute a difficult surgical emergency.

For this reason, it seems well, in the case of this and other common conditions, to review from time to time our own and others' experiences and to bring together what has been learned about the most effective methods of management. On looking back over the years during which I have been engaged in the practice of otolaryngology, there has been no outstanding innovation which marked a complete change in the methods of handling nasal bleeding, but there have been numerous modifications and improvements, as the result of various medical and surgical developments.

Before discussing these measures in more detail, it probably is well to enumerate the principal causes of hemorrhage from the nose. The local causes include: *1.* trauma, either accidental or surgical, or from picking, rubbing, and blowing the nose; *2.* acute and chronic infections; *3.* perforation of the septum with repeated hemorrhages from the denuded area; *4.* a spur or deflection of the nasal septum; *5.* neoplasms in the nose or nasopharynx, and *6.* hemorrhagic telangiectasis.

The general or systemic causes for bleeding from the nose are: *1.* hypertension and arterial changes, such as arterio-

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sclerosis; *2.* increased venous tension, as in emphysema, whooping cough, bronchitis, tumors of the neck or chest, aneurysms of the neck or chest; *3.* blood diseases, including leukemia, hemophilia, pseudo-hemophilia, multiple myeloma, pernicious anemia, secondary anemia, purpura, etc.; *4.* cardiac diseases, particularly mitral stenosis and rheumatic fever in children; *5.* a lowered prothrombin time due to hepatic disease or to the use of dicumarol or other anticoagulant drugs, acetyl salicylic acid, and the like; *6.* acute infectious diseases, especially at their onset; *7.* menstrual disturbances resulting in vicarious bleeding; *8.* scurvy and other types of avitaminosis, and *9.* high altitudes.

A relatively common site of posterior nasal bleeding in older persons is an area of dilated veins underneath the posterior end of the inferior turbinate. Woodruff has designated this area as the "naso-pharyngeal plexus." Bleeding from the lateral nasal wall is usually from the lateral branch of the sphenopalatine artery. The medial branch of this artery, sometimes called the nasopalatine artery, may also be the source of bleeding from the anterior inferior portion of the septum. Other types of nasal bleeding include that from behind septal spurs and deflections, diffuse bleeding from the nasal mucous membranes, usually seen in patients with blood dyscrasias, and bleeding from the anterior ethmoidal artery or vein, which is most frequently caused by trauma.

The incidence of epistaxis is highest in children but seldom is serious in these young individuals. The more severe forms of epistaxis are seen in older persons and are often related to the presence of degenerative vascular and other diseases. In a series of 212 cases of severe epistaxis requiring hospitalization, reported by Hallberg from the Mayo Clinic, the incidence increased directly with age. More than 40 per cent were over 60 years of age, and of this group, 65 per cent were from hypertension.

The multiplicity of causes for nasal bleeding and the various sites of hemorrhage make it obvious that the management of each case must be individualized. The severity ranges from a slight dripping of blood to profuse, shock-producing, and even fatal hemorrhage, and treatment must be applied accordingly. Whatever the cause or source of the hemorrhage, the aim of treatment is to stop the bleeding as promptly as possible. If the cause is purely local, the difficulty of the problem depends upon the site and type of vessel involved and the amount of blood lost. When the bleeding is secondary to systemic causes, thorough clinical studies and the collaboration of otolaryngologist and internist may be necessary before the problem is satisfactorily solved.

Therapeutic Armamentarium.—In the clinical management of epistaxis, a variety of coagulants are available for use according to the indications in the individual case. A variety of substances are used for nasal packing, for some of which special techniques have been recommended.[7,14,15,19,37] Besides these, there are the agents for chemical cautery and electrocautery, by the actual cautery or by electric coagulation. When medical measures and cautery do not suffice to control the bleeding, then ligation of the blood vessels is indicated. The surgical procedure most frequently employed is ligation of the external carotid artery; *in rare instances, interruption of the* external maxillary or one of the ethmoid arteries may be necessary, depending upon the site of the hemorrhage.[5,21,23,25,32,42,46,47,49,53,55,56,57]

Among the coagulants which have been recommended for use in the treatment of epistaxis are vitamin K,[31] which is necessary for the synthesis of prothrombin; protamine sulfate (Lilly), which is *an antidote for heparin*; vitamin C; Cortisone,[1,9,11,24,29,46] which has been shown to affect the coagulation of the blood and to exert a favorable effect in patients with *pseudohemophilia, or* prolonged prothrombin time; Koagamin (Chatham), which is an aqueous solution of oxalic and malonic acids, with 0.25 per cent phenol;[12] Neo-hemoplastin (Parke-Davis); snake venom, useful in capillary bleeding by direct effect *on clotting of fibrinogen; oxalic acid used in*tramuscularly or intravenously,[26] and pituitrin.[27]

The substances used for nasal packing, besides surgical gauze, include oxidized cellulose (Oxycel);[16,17,33,43] gel foam;[48] thrombin,[12,54] alone or in *combination with oxidized cellulose* or gel foam; Simpson splints; rubber balloons filled with air, water or vaseline gauze; sponge;[15] cotton, and salt pork (for the hypertensive patient).[10,38]

Substances used for chemical cautery are silver nitrate, trichloracetic acid, neutralized chromic acid, basic ferric sulfate or subsulfate (Monsel's solution), compound tincture of benzoin and burnt alum.[12,63]

Management of Epistaxis in Children.—It has often been noted that simple nose bleed will stop spontaneously, regardless of treatment used, or even without treatment. Although this is true, it must also be remembered that to the patient this fact may not be known or obvious. Whenever the bleeding from the nose is of sufficient severity that a physician is consulted, it is to the patient, or to his family, a serious matter and sometimes a frightening occurrence. For this reason, reassurance is the first step in treatment. If the patient is at home, instruction should be given that he should be placed in the proper position, that is, that he should sit up and bend forward. If he lies on his back, the stomach may fill with blood, causing faintness and nausea. In cases of simple nosebleed from the anterior septum, blowing out the clots and pressing the anterior nares together for a few minutes may be sufficient to control the condition.

Whenever intranasal manipulation or packing is needed, the administration of a sedative is indicated, whether the patient be a child or an adult. Once his anxiety is relieved and he becomes relaxed, whatever measures are necessary to control the bleeding are much more easily accomplished. In cases of anterior nasal bleeding from Kiesselbach's or Little's area, the bleeding vessel can frequently be seen, if the hemorrhage is not too profuse; then the area is cocainized and cauterized either with one of the chemical agents or the electric current. In attempting to control hemorrhage from the anterior part of the nose of a child, my associates and I prefer the use of chemical cautery to electrocoagulation. In many instances, we use a solution of 50 per cent silver nitrate which is immediately neutralized by sodium chloride solution and covered with holocaine and adrenalin ointment. Some authors suggest the use of a silver nitrate stick or chromic acid or trichloracetic acid;[2] the latter two should be neutralized by a solution of sodium bicarbonate.

When the hemorrhage in the anterior portion of the nose is so severe that it is impossible to find the bleeding point, the nose is packed with cotton pledgets saturated with cocaine and adrenalin until the hemorrhage ceases. Then an attempt is made to find the bleeding point in order that the area may be cauterized.

When the hemorrhage is from the anterior superior part of the nose, and especially when there is a history of recurrent or profuse bleeding, we have found it most satisfactory to use "spot" packing of oxidized cellulose or gel foam saturated with thrombin and held in place with a cotton sponge and a small piece of adhesive placed over the nostril to prevent the pack from being dislodged by sneezing. The cotton should be saturated with penicillin solution to reduce the chance of infection. We also administer an antibiotic systemically if the pack is to be left in place as long as 24 hours.

In case of recurrent nasal bleeding in children, which the parents usually say is spontaneous and frequently comes on at night, there is often a discharge with crusting of the septum near the anterior nares which may be due to infection or to allergy. The child with allergic rhinitis has a tendency to rub the nose to relieve the itching sensation and to pick at the crusts with the finger. Excessive drying of the nasal mucus greatly reduces its protective value and renders the capillary bed more accessible to trauma. A measure devised by Hilding and recommended by Hunnicutt[37] is helpful in preventing crusting and allowing the tissues to return to normal; this is to place a light plug of dry cotton in the nostril for a few minutes several times a day. In cases in which any nasal discharge is present, it is important that appropriate treatment for the underlying condition be instituted.

An anterior deviation of the nasal septum is frequently a contributory cause in recurrent epistaxis, since it increases the likelihood of trauma of the convex side, and often there is also an accumulation of mucus with crust formation on the side of the convexity.

In all instances of continued or recurrent epistaxis in children, a thorough clinical examination and appropriate laboratory studies are indicated, to determine whether the bleeding is being caused by some systemic disease. Nasal hemorrhage of systemic origin in children is frequently noted in diseases of the blood, liver or kidneys, and in acute infectious diseases, particularly in acute rheumatic fever. In such instances, treatment of the underlying disease is of paramount importance, with the local measures which are necessary for the control of bleeding from the nose.

Kugelmass[44] reported that nontraumatic, nonirritant nosebleed occurred in about one-third of rheumatic children observed over a 15-year period, especially at the onset of acute episodes. This hemorrhage is attributed to increased vascular fragility of the nasal mucous membranes, which is said to be pronounced during puberty and prevalent during active carditis. He reported favorable effects in such cases from the use of vitamin P (rutin) derived from lemon rind and administered as tablets of eriodictyol or capsules of the chalcone. Kugelmass said that the medication increased capillary resistance above the critical level for bleeding but not to the normal level, and was effective in diminishing the frequency and severity of the rheumatic epistaxis.

Since Cortisone has been reported to have a favorable effect during the acute attack of rheumatic fever[34] which is the time that epistaxis is usually most pronounced, and since it also apparently increases the coagulability of the blood,[11] it is to be expected that its administration during the acute phases of rheumatic fever may have an influence in controlling the epistaxis observed in this condition.

When the history, general physical examination and laboratory findings indicate that the nasal bleeding is not due to any systemic disease, we believe it useful to prescribe large daily doses of vitamin K and of vitamin C and to increase the quantity of fruit juices, in the diet, even though there is no evidence of clinical nutritional deficiency, and the diet is thought to be adequate.[27,51]

Treatment of Epistaxis in Adults.—The type of nasal hemorrhage seen in children may also be seen in mature individuals, although with considerably less frequency. The milder forms of epistaxis in adults are treated as they are in children. The more severe episodes of hemorrhage from the nose are encountered, as a rule, in elderly individuals with hypertension and other degenerative diseases. The bleeding in such cases is often from the upper or posterior areas, where it is difficult to find the eroded vessel.

Quite frequently, patients with severe epistaxis have been bleeding profusely for several hours, and sometimes for several days, before they are seen by the otolaryngologist. For this reason, it often is necessary to administer tranfusions or other countermeasures against shock before the local situation can be dealt with adequately. It must be remembered, too, that sometimes the patient who presents a shock-like appearance is merely nauseated from swallowing too much blood. If there is any uncertainty as to the patient's general condition, however, transfusion of 500 ml. (average) of blood should be given.

When the bleeding is still active, any packing that has been placed in the nose is removed and the nose is cleansed with a suction apparatus under direct vision in attempting to find the source of the hemorrhage. For this purpose, I have found the Anthony-Fisher suction-cautery a very useful in ment. Cotton pledgets soaked in adrenalin and co used to shrink the mucous membrane and provide anesth for manipulation.

On some occasions in my experience, when using the tion apparatus in cases of this type, the nasal hemo encountered was so profuse that it was impossible to the blood quickly enough to determine the area from wh originated. In such instances, insertion of a post-nasal followed by firm packing of the nose, was the only that could be carried out.

One-inch gauze, carefully and firmly applied in laye the material most often used for packing the anterior po of the nose. Hallberg[27] suggests that the gauze be covered with petrolatum. Others have recommended the packing material in peroxide[7] or in tannic acid and pyrine solution.[41] In our work, we prefer to use a gauze oratory studies are indicated, to determine whether the bleeding is being caused by some systemic disease. Nasal hemorrhage of systemic origin in children is frequently noted in diseases of the blood, liver or kidneys, and in acute infectious diseases, particularly in acute rheumatic fever. In such instances, treatment of the underlying disease is of paramount importance, with the local measures which are necessary for the control of bleeding from the nose.

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When the bleeding is still active, any packing that has been placed in the nose is removed and the nose is cleansed with a suction apparatus under direct vision in attempting to find the source of the hemorrhage. For this purpose, I have found the Anthony-Fisher suction-cautery a very useful instrument. Cotton pledgets soaked in adrenalin and cocaine are used to shrink the mucous membrane and provide anesthesia for manipulation.

On some occasions in my experience, when using the suction apparatus in cases of this type, the nasal hemorrhage encountered was so profuse that it was impossible to remove the blood quickly enough to determine the area from which it originated. In such instances, insertion of a post-nasal pack, followed by firm packing of the nose, was the only procedure that could be carried out.

One-inch gauze, carefully and firmly applied in layers, is the material most often used for packing the anterior portion of the nose. Hallberg[27] suggests that the gauze be heavily covered with petrolatum. Others have recommended soaking the packing material in peroxide[7] or in tannic acid and antipyrine solution.[41] In our work, we prefer to use a gauze packing with 5 per cent sulfathiazole and to cover the post-nasal pack with 5 per cent sulfathiazole ointment. Penicillin or other appropriate antibiotic is administered while the pack is in place, to prevent infection.

With the use of antibiotics and the sulfonamides, packing may be kept in the nose for a week or longer, without danger of infection. If the bleeding is completely arrested by the packing procedure, sufficient healing takes place so that the bleeding point is permanently sealed, and, when the packing is removed, the nasal mucous membrane usually appears to be in very good condition. In our experience, the instances in which gauze packing was left in the nose for a week, or thereabouts, were those in which severe bleeding had occurred for a protracted period before we saw the patient, and it was deemed advisable to restore blood volume and electrolyte balance and to administer vitamins before any attempt should be made to find the bleeding vessels.

There are numerous instances when the Foley catheter, the Anthony-Fisher post-nasal air balloon or the Cooper Rose inflation plug may be preferable to use of a post-nasal tampon and nasal packing with gauze or sponge. Numerous other packing materials have also been suggested. Dixon[15] has advocated the use of pliable marine sponges, applied piecemeal, for the control of epistaxis. Packing with salt pork[10,58] for the control of bleeding in the hypertensive patient has been widely recommended in the literature, but I have never been impressed with the value of this measure. I tried it on numerous occasions, but have long since discontinued any attempt to use it. I found that these plugs were difficult to keep in place and very often did not control the hemorrhage.

During the past few years, there has been considerable interest in the use of some of the newer hemostatic agents, such as oxidized cellulose,[16,17,35,43] fibrinfoam,[4,18,58] and gel foam[45] for packing in the case of severe nasal hemorrhage. Houser[35] recommended using a strip of oxycellulose ½-inch wide in sufficient quantity to exert the necessary pressure for control and stated that the gelatinous mass could be removed at the end of 24 hours, or that when used with a post-nasal pack, the latter may be removed in 24 hours, while the absorbable cellulose is left *in situ*.

On the basis of my own experience, my opinion is that neither oxidized cellulose (Oxycel) nor gel foam should be used in the nose in large amounts, such as in the control of severe hemorrhage or following submucous resection. One unhappy experience resulted in a slight injury to the mucous membrance when I was attempting to remove the jelly-like mass by means of suction and forceps. The patient now has a moderately large perforation on the septum which she is unable to forget, because of the crusts that tend to form. On the other hand, when the Oxycel or gel foam may be used in small quantities for "spot" packing or in situations where the packing may be applied without completely obstructing the nasal passage, it is quite useful. A small strip of Oxycel or gel foam saturated with thrombin is useful in controlling the bleeding following the removal of nasal polyps. In this instance, however, if the packing is not loosely applied, the patient will complain of severe headache that is most difficult to control.

Thrombin[13,14] has proved itself useful for sealing the mucous membrane flaps following submucous resections. This is accomplished by spraying a small amount of the liquid thrombin between the flaps and gently pressing them together; in most instances, this procedure is sufficient to control the bleeding. It should be emphasized that thrombin is never injected, but is always used as a topical application for the control of hemorrhage from small superficial vessels. If bleeding should occur following a submucous resection, it is well to use Simpson splints or rubber finger cots packed with vaseline gauze.

In instances in which the bleeding point cannot be located and/or cannot be cauterized satisfactorily, or in those in which bleeding recurs following nasal packing, then additional local manipulation, systemic treatment, and appropriate procedures, which may include ligation of a principal artery, must be carried out, according to the site of the hemorrhage and the indications in the individual case.

A procedure that has been recommended[3,22,50] in some cases in which routine cauterization is not successful is the injection of sclerosing solutions underneath the mucosa around the bleeding point. Among the solutions used for this purpose are 0.5 milliliter of 10 per cent phenol in almond oil; Sylnasol, 0.25 to 0.5 milliliter, and 0.1 milliliter of a saturated (16.6 per cent) solution of quinine lactate.

The epistaxis due to telangiectasia (Osler's disease) presents a special problem in treatment.[20,41,60] There may be multiple bleeding points from angiomas situated in the nasal mucous membranes. Packing may aggravate the bleeding because of injury to the mucous membranes. As a temporary measure, most authorities agree that the rubber balloon is *most satisfactory in controlling the hemorrhage in this type* of case. The local lesions may be treated by electrocoagulation, which is preferable to the actual cautery or chemical corrosive agents. A combination of beta radiation and deep Roentgen-ray therapy has been advocated for treatment of the local lesions by Crowe and his associates at Johns Hopkins. Repeated blood transfusions frequently are necessary in this condition to combat the anemia resulting from the frequent hemorrhages.

In certain blood dyscrasias, such as acute leukemia and multiple myeloma, there may be diffuse bleeding from the mucous membrane, including the nasal mucosa. In these cases, topical application of thrombin and thromboplastin is often useful. Since packing traumatizes the membranes, this is not resorted to unless the bleeding is so severe that it is necessary in order to save life.[27]

Cortisone has been used with favorable effect in idiopathic thrombocytopenic purpura,[1,34] allergic purpura,[1] chronic hypoplastic anemia[34] and pseudohemophilia.[39] Epistaxis is a frequent *manifestation* in *these blood* dyscrasies, and has responded, along with other hemorrhagic symptoms, to the administration of cortisone or ACTH.

Kirschner[42] called special attention to the bleeding which may be caused by polyps broadly attached to the anterior portion of the septum, and outlined the following procedure:

"The base is cocainized and separated from its subjacent layer by gauze pledgets soaked in stryphnon or liquor ferri. These are left in place over the site of the hemorrhage until a conglomeration forms. Pressure is applied from the external nostril and after a few minutes, the conglomerate can be removed. Further procedures are not as a rule necessary." Kirschner also cautioned that galvanocautery and strong caustics should not be used in the poorly nourished anterior portion of the septum, since they involve danger of perforation.

Hunnicutt[34] has recommended crushing the turbinate over its entire length as an efficient method of stopping the bleeding from an artery going to the inferior turbinate. The artery is at the surface on the lateral and inferior aspect just as it enters the turbinate at its posterior end, and it is at this point that the crushing obliterates the vessel.

Septal bleeding behind spurs and deflections may require a submucous resection as preliminary treatment before the bleeding point can be found. In numerous instances, the submucous resection alone is sufficient to control the hemorrhage. For patients who have anterior septal perforations with crusting and repeated bleeding, Hallberg[27] recommends a modified submucous resection with removal of exposed cartilage around the edges as the treatment of choice. Beinfield[6] lists the theoretical reasons why submucous resection may control nasal hemorrhage as follows: *1.* Bleeding not controlled by a postnasal pack may arise from the dilated plexus of veins underneath the posterior end of the inferior turbinate[41] and may be stopped by elevation of the mucous membrane during a submucous resection; *2.* It may be that infiltration of the anesthetic raises the mucoperiosteum of the septum, sphenoid and floor of the nose and temporarily produces pressure upon the vessels, long enough to form a clot, in addition to the vasoconstrictor action of the epinephrine in the solution; *3.* Postoperative tissue reaction to the infiltration and surgery may produce edema that constricts the vessels by its pressure; *4.* Elevation of the mucous membrane from the septum may change the position of the bleeding vessel, permitting it to retract or thrombose; *5.* The elevated septal flaps may alter the relationship of the sphenopalatine arteries to the rigid resected bony septum when the flaps heal together, giving the arterio-sclerotic vessel an added advantage of mobility to retract with whatever slight elasticity it may still have.

When the medical measures and minor surgical procedures already outlined fail to control severe nasal bleeding, then recourse must be had to ligation of one of the arteries supplying the nose, above the bleeding point. The use of ligation of a main artery for the control of epistaxis is not new, nor are the surgical procedures specific for this purpose. The incisions and techniques used are the same as those employed in operation on the sinuses or neck for other reasons, such as the eradication of infectious or malignant lesions.

Although in the earlier literature there were numerous reports of ligation of the common carotid or internal carotid arteries for nasal hemorrhage, this is a formidable and often unsuccessful procedure. The ligation which is most often indicated and performed for intractable hemorrhage from the posterior portion of the nose is that of the external carotid artery.

Although it is more than probable that most of the ligations of the external carotid artery which have been carried out for treatment of epistaxis have gone unreported there have been several reports which dealt with this subject. When McKnight[49] reviewed the literature on carotid ligation in 1926, he found 17 instances in which ligation of the external carotid artery for nasal hemorrhage had been reported. Spar and Hallberg[57] noted eight additional cases in the literature and reported a series of 11 cases, observed in an 18-year period at the Mayo Clinic, in which external carotid ligation was done for nasal hemorrhage. In a later report of 212 cases of severe epistaxis, Hallberg[27] mentioned that 17 had required external carotid ligation. Another case of ligation of the external carotid artery for nasal hemorrhage was reported by Fitz-Hugh and Risher.[21]

Ligation of the internal maxillary artery for severe hemorrhage from the sphenopalatine artery was advocated by Seiffert,[42] as being more effective than occlusion of the carotid artery, which displays anastomoses which may keep the circulation in motion in the bleeding area. His technique included removal of the facial wall of the antrum of Highmore, chiseling out a large section of its posterior wall which was removed with a hook, after which the periosteum was cleft, and the artery isolated near its point of exit from the sphenopalatine foramen, and ligated. Hirsch,[32] in 1936, reported a case in which this approach was used. At about the same time, Sewall[63] mentioned that he had used the transantral approach for ligation of the internal maxillary artery in two instances of profuse bleeding. Kirschner[42] commented that ligation of the internal maxillary artery and other terminal branches of the external carotid is rarely necessary. Although the internal maxillary artery may be the source of profuse bleeding, particularly in some cases of trauma, such hemorrhage is usually dealt with by ligation of the external carotid.

During recent years, there have been several reports of ligation of the anterior ethmoid artery for nasal hemorrhage. Goodyear,[26] who reported a case in 1937, is usually credited with priority in the ligation of the anterior ethmoid for epistaxis, but this was an essential feature of the fronto-ethmosphenoid operation, which was performed with relative frequency in the decades preceding Goodyear's report. Luongo,[46] Sewall,[75] and Simpson[64] all mentioned the ligation of the anterior ethmoid artery (and the other ethmoid arteries) as applicable to the control of obstinate nasal hemorrhage.

The incision of the ethmoid ligation is made along the eyebrow. Luongo described the elevation of the periosteum as being started at the upper third of the incision with downward sweeping movements, followed up by forward ones. The anterior ethmoidal artery is usually found in the orbit within 3 cm. It is tied and severed at the foramen. The posterior ethmoid artery, in case it should be the source of hemorrhage, is found a short distance behind the anterior vessel.

Besides Goodyear, others who have reported ligation of the anterior ethmoid artery for severe epistaxis are Macbeth,[47] Oppenheim, *et al.*,[53] who reported two cases, and Fitz-Hugh and Risher.[21] In the case described by the latter authors, ligation of the external carotid artery failed to control the hemorrhage, and the anterior ethmoid also had to be interrupted before the bleeding ceased completely.

The foregoing outline of the instruments, agents and procedures available for the control of nasal hemorrhage serves to demonstrate that there is no one routine or best treatment for this condition, which arises from multiple causes and exhibits a wide range of severity. Each case has to be treated according to the indications presented, and the purpose in every instance is to control the bleeding as promptly as possible with the most conservative means that may prove effective.

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MEETING OF THE GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The Georgia Society of Ophthalmology and Otolaryngology will meet March 11-12, 1955, at the General Oglethorpe Hotel, Savannah, Georgia.

The speakers will be: Dr. Francis H. Adler, Philadelphia; J. W. McCall, Cleveland; Dr. J. A. Hilger, St. Paul; Dr. Walter H. Fink, Minneapolis; Dr. James H. Allen, New Orleans; and Dr. P. E. Ireland, Toronto, Canada. For further details, address ALTON V. HALLUM, M.D., Secretary, 245 Doctors Bldg., Atlanta 3, G[illegible]

NORMAL HEARING AND ITS RELATION TO AUDIOMETRY.

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INTRODUCTION.

The advantages of audiometry over other methods of examining threshold auditory acuity are such that in many countries the audiometrist is becoming an indispensable aid to the otologist; yet it is clear that much of the advantage is lost unless certain standards can be maintained as to the characteristics of the audiometer and the way it is used. In this country, something has been done in standardizing the instrument,[1,2] though little has been standardized in the way it is used. In many other countries, likewise, national bodies have written specifications for the instrument. So far as the writer is aware, no other country has adopted more detailed specifications than contained in the reference cited above.

Many features of an audiometer must be standardized, but none of more fundamental importance than the frequencies used and the intensity level of the acoustic output which describes "Normal Hearing." There is by now a fairly general use of the octaves from 125 to 8000 cps as the frequencies to be used, but a disagreement exists as to just what intensity, at any particular frequency, shall describe "Normal Hearing."

American audiometers set "Normal Hearing" in accordance with values established by the U. S. Public Health Service and on record in the National Bureau of Standards (NBS).[3] Almost from the time these values were established, however, dissatisfaction has been expressed. Bunch,[5] for example, with probably the most extensive clinical experience in the country, set his own "Normal" at intensities up to 10 db fainter than the present standard at the higher frequencies. It is well known that the average hearing of those in the age group

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20 - 29, tested at the 1939 New York World's Fair[22] was, at 3520 and 7040 cps, up to 10 db *better* than the same age group tested by the Public Health Service Survey of 1935 - 36 —even though the World's Fair study included some ears known to be seriously defective. Lüscher and Zwislocki[11] stated that their American audiometers yielded values, on supposedly normal-hearing individuals, of 10 and 15 db fainter than our American standard. The British[6,26] have recently found thresholds for normal-hearing individuals which they believed to be considerably fainter than our standard. It seems evident that a re-examination of the standard is called for.

Of course, so long as one can calculate from audiometric data an individual's hearing threshold, for the equipment used, in terms of the international reference intensity (10^{-16} watt/cm,^{2} equivalent to a sound pressure level (SPL) of .0002 dyne/cm^2), it is for scientific purposes immaterial just what intensity level is produced by "zero" on the audiometer. For example, it can be calculated that a 20 db hearing loss on an American audiometer is equivalent to a threshold of 37 db above reference SPL in a certain standardized closed coupler, and the same figure could still be derived no matter where the audiometer zero was maintained; however, the reporting of audiograms in terms of threshold SPL does not have the merit of simplicity, and furthermore is quite unnecessary if agreement can be reached internationally on just what "Normal Hearing" (*i.e.*, audiometric zero) should be.

1. History of American "Normal Hearing."

Accounts of the development of the audiometer in this country and abroad have been given by Bunch,[5] and of the fixing of American "Normal Hearing" by Watson and Tolan.[23] Only those features pertinent to the present question will be detailed here.

The first really successful commercial audiometer in this country was the Western Electric 2A. It was calibrated at the Bell Telephone Laboratories, "Normal Hearing" being taken as the average dial setting at threshold of otoscopically normal observers between 20 and 30 years old, the number variously given as 36[20(p. 270)] or 41.[14(Bull. 4, p. 1)] Earphones used were the W.E. Type 552. Similar equipment was used in the Public Health Survey in 1935 - 36. In that survey, careful physical calibration was maintained among the 17 different 2A audiometers, and the 31 audiometricians exercised the correct precautions looking toward reliable data. Subjects were volunteers but, of course, inexperienced in psycho-physical judgments.

Data were averaged from both ears of 1242 persons, aged eight to 76 years, normal by clinical examination, with history of no loss for speech in either ear, and whose air conduction audiograms for both ears did not exceed a variation of 20 db. It is this group, then upon which American "Normal Hearing" depends.[14 (Bull. 4, p. 3)]

The average thresholds at the eight test octaves from 64 to 8192 cps for this group were found in terms of dial settings on the W.E. 2A audiometer. The average threshold voltage to the ear phone at each frequency was found to be as follows:[1]

cps	Db below 1 volt
128	73
256	88
512	102
1024	109
2048	111
4096	107
8192	75

Shortly after this information was available, a much improved earphone was produced, the W.E. Type 705A. Three of the most stable phones of this type were compared with the standard Type 552 units, by loudness balancing using six observers and three loudnesses (threshold, 20, and 40 db over threshold). The voltages at the 705A terminals were thus found which produced the equivalent threshold SPL of the Type 552 phones.

The development of the Type 9A 6cc brass coupler at NBS provided a way to pass from earphone voltage as a standard, to acoustic output in a standard cavity the volume of which has an impedance approximately equal to the equivalent volume of the ear. This includes the volume behind the tympanic membrane. The 705A earphone is placed at one end of the cylindrical cavity, and a sensitive microphone, the W.E. 605AA, calibrated in dynes/cm,^{2} is placed at the other end. Proper acoustic seals and precautions against vibration are maintained. Threshold voltage is then applied to the terminals (or usually 60 db above threshold voltage), and the SPL in the cavity is read with the microphone. By this technique the NBS is able to state "Normal Hearing" as the acuity of an observer whose threshold is reached when the acoustic output of the W.E. 705A phone is such as to produce the following SPL in the 9A coupler:[2,3]

cps	SPL in db re 0002 dyne/cm^2	
125	54.5	
250	39.6	Note. Original data were at octaves of 128; data for octaves of 125 were obtained by graphic interpolation.
500	24.8	
1000	16.7	
2000	17.0	
4000	15.1	
8000	20.9	

The great advantage of this standard is that a laboratory need not actually have on hand one of the three standard 705A earphones, now reposited at the NBS, with which to compare its own equipment, but can utilize a 9A coupler to transfer standards to his own 705A phone, and thence, by loudness-balancing, to any other earphone, or earphone-cushion combination, for which the 9A coupler is appropriate.

Loudness balancing has recently been performed by the NBS[13] between their 705A units and two other types of earphones now in common use with audiometers. From these data has been derived the SPL in the 9A coupler which produces equivalent threshold loudness with the 705A standards:

cps	Permoflux PDR-1 in MX-41/AR Cushion	Permoflux PDR-8, in MX-41/AR	W.E. 705A
125	51.3	52.4	54.5
250	40.1	38.8	39.6
500	25.6	24.5	24.8
1000	17.8	16.5	16.7
2000	17.5	17.1	17.0
4000	11.6	14.5	15.1
8000	28.1	28.3	20.9

2. What is the best method of specifying "Normal Hearing?"

Standardizing "Normal Hearing" in a closed acoustic coupler, as outlined above, is an immensely valuable technique for practical reasons, and it would be all that would ever be required if the coupler perfectly simulated the human ear, but unfortunately the 9A coupler does not perfectly simulate even an average human ear; furthermore, the outputs of the phones commonly used on present-day audiometers vary with variations in the acoustic impedances of individual ears. Thus, a phone which generates a certain SPL in the 9A coupler, may generate different levels when coupled to human ears.

Wiener and Filler[27] dramatically showed this when they impressed constant voltage upon an earphone, and mounted it successively on 11 men. Sound pressure level was measured by a small probe tube inserted under the earphone cushion and led to a microphone. Now if the earphone generated the same SPL for all ears, the measurements would show a negligible variation; but, as a matter of fact, the measurements differed from man to man by as much as 10 db at many frequencies, and up to 20 db at the extremes.

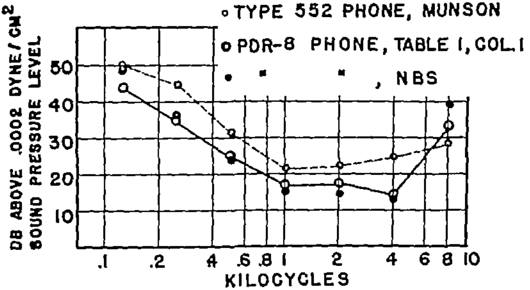
This result illustrates the danger of measuring threshold pressures in a transfer coupler rather than in the ear canal itself: let us take two men, in one of whose canals the earphone is 10 db more efficient than in the other's; at threshold, their earphone voltages (and, therefore, SPL in the 9A coupler) will differ by 10 db, yet in reality their thresholds may be identical, in terms of SPL at the eardrums. Here the threshold by coupler SPL is an erroneous datum. Only by probe technique could the true state of affairs become known.

The practical feasibility of probe tube technique was accentuated by Filler, Ross, and Wiener,[8] who found that results were not affected by "drastic" bending of the flexible tube, by "small" variations in length, or by angle of incidence of sound between 200 to 8000 cps.

The writer concludes that for the careful determination of SPL at an individual's threshold, a direct reading of threshold SPL by probe tube is preferable to an inferred reading with the intermediary of the brass coupler.

3. What is "Normal Hearing" in terms of ear canal SPL?

Standardization groups in this country have not as yet specified "Normal Hearing" in terms of anything but the 9A coupler. It would, however, be of great practical importance if it were possible to state what SPL *measured in the ear canal* actually constitutes a "Normal Hearing" standard. In this way one would avoid all the problems connected with discrepancies between the 9A or any coupler and the human ear, discrepancies between couplers, the necessity to construct a new coupler for every new type of earphone or earphone-cushion combination, and all the problems concerned with loudness-balancing among earphones, etc.



ESTIMATES OF EAR CANAL SPL AT AMERICAN "NORMAL"

FIG. 1

Munson[10] has provided a bridge by which we may pass from "Normal Hearing" on American audiometers, to SPL in the ear canal. On a "small number of people" he set up "Normal Hearing" on the 2A audiometer and measured SPL by probe "at the opening of the ear canal." In this work, note that the subjects were used only as passive couplers between earphone and microphone. We have, now, only to add to these figures the corrections made to the 2A "Normal" points by the Public Health Survey in order to obtain the SPL at the opening of the ear canal by NBS standards. Munson's data, corrected as described, are in Fig. 1, and represent American "Normal Hearing" in terms of SPL under the 2A earphone (it must be cautioned that these figures are not necessarily correct for other phones or phone-cushion combinations).

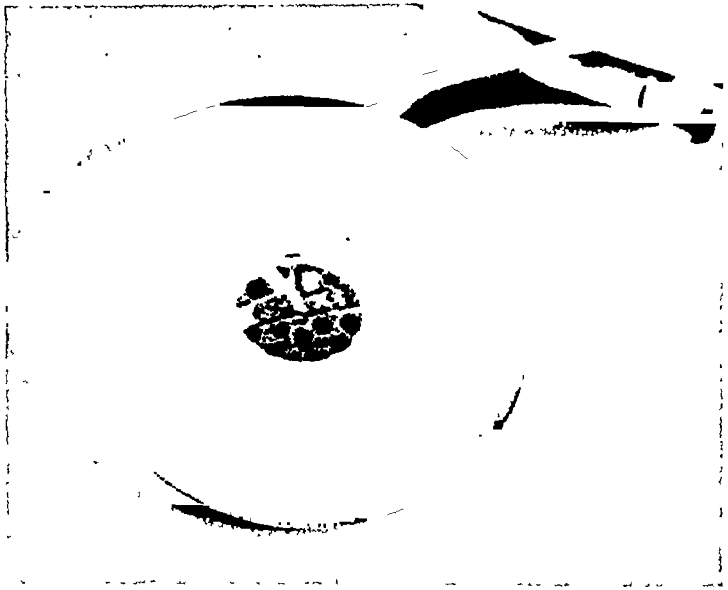


Fig. 2. Experimental Phone, Permoflux PDR-10 with Probe Tube Affixed.

Another and rather different estimate of canal SPL at American "Normal" is available in a recent preliminary report[15] from the NBS: using 16 ears as passive couplers just as Munson did, a constant voltage was impressed on the W.E. 705A phone and also on a Permoflux phone, Type PDR-8. For both phones, SPLs developed in the ear canals and (at the same voltage) in he 9A coupler w -ported for head-band forces of 1000. 1500, and 2000 gr

The writer computed the difference - NBS study between the coupler SPL and the canal S nd used this difference in Fig. 1 to estimate canal SP American "Normal Hearing." Data for the PDR-8 p only is entered,

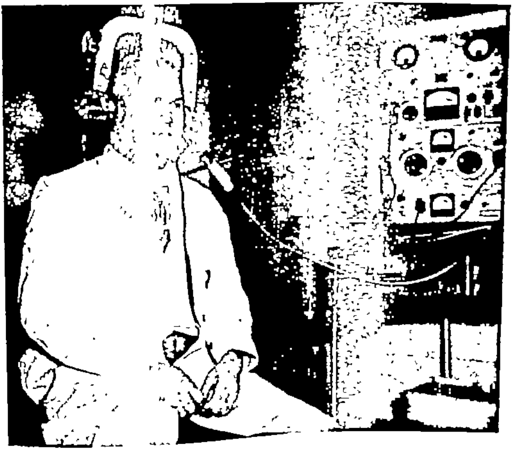


Fig. 1. View of Phone Holder, Probe Tube Microphone, and Sound Pressure Level Measuring Equipment.

the 705A phone yielding very similar though not identical results. At 8000 cps, for example, the difference between the 705A and the PDR-8 earphones amounted to as much as 13 db.

The discrepancies in Fig. 1 between Munson's estimate and that of the NBS led the writer to resolve the problem by still a third estimate. A Permoflux PDR-10 earphone was fitted with an MX-41/AR cushion (see Fig. 2) and mounted in a plastic helmet (see Fig. 3) with a calibrated pneumatic system providing a force of 2000 grams against the head. A probe tube 8.8 cm long, o.d. 1.9 mm. i.d. 1.2 mm. was fastened to the phone as in Fig. 2. Its tip looked directly into the meatus, and protruded 11 mm from the face of the earphone. It was led to a small brass tube and attached face plate for screwing onto a 640AA microphone exactly as specified by Nichols, *et al.*[16] and as re-published by Beranek.[4]

This system was connected to a calibrated W. E. 640AA microphone and to the Western Electro-Acoustic Company's pre-amplifier, octave-band filter, and voltmeter calibrated in dynes per cm².*

TABLE I.

SOUND PRESSURE LEVEL IN EAR CANAL AT AMERICAN "NORMAL HEARING," FOR THE PERMOFLUX PDR-8 PHONE IN THE MX/41/AR COUPLER (Data Taken by Probe Tube under an Experimental Phone, and Transferred to the PDR-8 Phone by Loudness Balancing).

cps	Median	Mean	Standard Deviation	PDR-8 is More Efficient Than Experimental Phone by:
125	44.3	45.5	6.51	3.7 db
250	35.0	33.9	4.47	1.8
500	25.2	25.1	1.63	–0.6
1000	17.2	17.3	1.31	–0.3
2000	17.6	17.1	2.48	0
4000	14.3	13.9	2.68	1.4
8000	33.4	33.6	5.90	–2.9

The probe tube was calibrated by the writer in the 9A coupler, the tube tip entering the cavity in three ways in succession: (a) connected to the earphone as described above, and, therefore, entering the cavity from above; (b) entering the side of the cavity; (c) through a hole in the middle of a brass plug which replaced the microphone at the bottom of the cavity for this measurement. A second independent measurement at all three positions was completed, and a mean of *all six readings was computed for each audiometer frequency.* The range of variation among the six readings was never as much as 4 db at any frequency, and the mean of the two readings at any position never differed by as much as 1 db from the final mean adopted, except at 4000 where it amounted to 1.8 db for one position.

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* The maintenance of this equipment was in the daily charge of Mr. C. E. White, E.E.

When this equipment was assembled and calibrated, the first 50 men reporting to this laboratory for hearing testing were fitted with the helmet, a force of 2000 grams was applied, and voltage applied to the phone which generated "Normal Hearing" SPL in our 9A coupler. Canal SPL was then read.

The results are in Table I, and the median reading is entered in Fig. 1. Our data are seen to confirm the NBS report and are used in the rest of this paper as an estimate of American "Normal Hearing" in terms of ear canal SPL.

4. Is this inferred canal SPL confirmed by hearing surveys with probe tube technique?

While Table I can be thought of as a standard for canal SPL at "Normal Hearing," it must be remembered that it is based upon the original Public Health Survey, with which some dissatisfaction has often been expressed. What one wishes to know, now, is whether the Public Health Survey definition of "Normal Hearing," as expressed in Table I, agrees with the results of other surveys, by probe tube, of the hearing of clinically normal persons.

Sivian,[10] in 1928, was the first to use a probe tube, inserted about half-way into the meatus to a point 1-1.5 cm from the eardrum. He determined thresholds at frequencies from 500 to 15,000 cps on eight ears, using two procedures: (a) the ear open and stimulated by loudspeaker, and (b) the ear under an earphone, the tones matched in loudness with the free field situation. SPL was measured by probe tube in both cases. Agreement was said to be good. Sivian's thresholds however, are rather less acute than others subsequently reported, and have generally been disregarded as coming from too small a population.

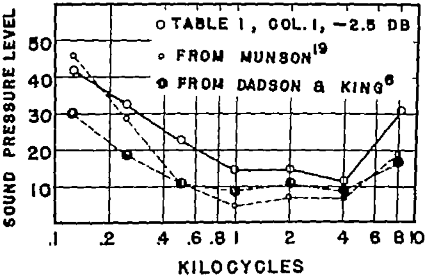
Munson,[11] in 1932, also used the probe tube to study 22 ears from 62 to 15,000 cps. Up to 2500 cps, the tube was inserted just under the earphone cushion; at higher frequencies the earphone calibration was obtained from Sivian's work, at 1-1.5 *cm* from the eardrum, . . . "on the assumption that equality of loudness of the tones from the loudspeaker and from the receiver, represent equal pressures on the drum." Later work[12,13,17,18] has shown this to be true for the frequencies involved here.

Munson's work is especially significant since it forms the major basis for the curve accepted by the American Standards Association as Minimum Audible Pressure. This curve, taken directly from the paper of Sivian and White[19] is at frequencies above 250 cps largely a slight smoothing of Munson's data (except for a 10 db discrepancy at 12 kc.).

At 250 cps and below, Sivian and White gave greater weight to the work of Wegel, *et al.*[23] who used insert earphones enclosing about 1 cc, and calibrated on a 1 cc. coupler, and the data of Fletcher and Wegel[9] who calibrated their earphones by loudness balance against a thermophone calibrated in a 1 cc. coupler. Reasoning from more recent work on the divergences of coupler SPL from eardrum SPL at lower frequencies, the writer believes that Munson's data form a more nearly correct picture of Minimum Audible Pressure than the composite curve of Sivian and White. Accordingly, Munson's data are entered in Fig. 4 as the best estimate in 1932 of canal SPL for normal hearing.

The next reasonably large surveys of canal SPL for normal hearing were performed by British teams, Dadson and King,[4] and Wheeler and Dickson,[26] Dadson and King have recently found, on 99 otologically normal men and women aged 18-25 years, thresholds which agree substantially with Munson. They regard it as unjustified to include older ages in a study of normal hearing since one thereby deliberately includes the hearing defects which accompany advancing age. There is good reason for this point of view. As they state, "It seems natural to regard the primary function of a pure tone audiometer as being the comparison of the hearing of a subject with a standard representing hearing which is free from impairment, and it thus seems more satisfactory to adopt as a standard of reference a group of subjects whose auditory state is the most likely to be free from impairment of any kind." (p. 387)

Fig. 4 reproduces their mean of two readings of threshold SPL by probe tube. It can be seen that their data are of the order of magnitude of Munson's data except at 125 and 250 cps, where the British thresholds are respectively about 18 and 10 db fainter.



NOTE: BRITISH DATA FALL 3–14 DB FAINTER THAN AMER. "NORMAL"

COMPARING EAR CANAL SPL AT AMERICAN "NORMAL" WITH TWO PROBE TUBE SURVEYS

FIG. 4

The study of Wheeler and Dickson, using similar equipment and identical calibration facilities, reported closed-coupler data at threshold on 514 men aged 18-23 years, clinically normal, intelligent, and able to pass a whisper and tuning fork test. A clinical audiogram was obtained but was not used to select the population further, and the clinical audiogram was not reported. The technique and cautions used seem without question to have been impeccable. The data reported can be directly compared with Dadson and King's data; not, it is true, with their canal SPL but with their closed-coupler data which correspond to their canal SPL at threshold. The data of Wheeler and Dickson on this basis differ from those of Dadson and King by only 2 db at the most (mean values at 2000 cps), and usually by much less, and are, therefore, not graphed in Fig. 4. This large survey lends considerable support to the figures for canal SPL at threshold for normal-hearing ears as reported by Dadson and King.

For comparison, American "Normal Hearing" in terms of canal SPL has been entered, minus 2.5 db to allow for the fact that relatively coarse intensity steps were used in the Public Health Survey.

We have collected in Fig. 4, now, three estimates of canal SPL at normal-hearing threshold: *1.* American "Normal Hearing" expressed in those terms, *2.* Munson's 1932 survey of 22 ears, and *3.* Dadson and King's 1952 survey of 198 ears. The differences between the American standard and the two laboratory surveys are clear, and a further difference between the laboratory studies appears, as noted earlier, at 125 and 250 cps. Evidently it would be impossible, on the basis of these data alone, to fix upon any international standards in audiometry.

It seemed in order to conduct another laboratory study in this country along lines of the British work, to help resolve apparent discrepancies. The present paper is one such study.

SUBJECTS.

In this work, the selection and number of subjects can be vital. Our subjects were men 17-23 years of age, physically healthy, with no service-connected auditory trauma, and with an intelligence quotient of 100 or better. None knew of any family-connected hearing disability or had any history of severe ear infections or operations. All were normal by whisper tests. One man only, of a series of 51 consecutive men reporting to this laboratory for routine hearing testing, was rejected from this study on the grounds that he had a hearing loss of more than 15 db at two or more audiometer frequencies. The remaining 50 men were accepted, and data on their left ears form the basis of this study. Testing was completed within two weeks after starting the study. The assistance of Mr. C. K. Myers, who collected data on nine of the men, is gratefully acknowledged.

EQUIPMENT AND PROCEDURE.

A Maico E-2 audiometer was modified in two ways: *1.* The interrupter circuit was entered and external leads provided which made and broke the circuit by means of a rotating disc, photocell, and relay in such a way that the tone was on for one second (counting from the point at which 100 per cent intensity was reached), and off for one second. *2.* The output of one channel was led to an impedance-matching transformer and to a bank of attenuators in 10, 1, and 0.1 db steps. Another impedance-matching transformer was inserted, and the output finally led to the Permoflux PDR-10 phone in the cushion and helmet shown in Fig. 3.

The subject was seated comfortably, the helmet and earphone adjusted, and 2000 grams force applied. The subject was given instructions to raise his index finger all the while a tone was heard, and drop it otherwise. Practice was given at 500 cps with the intensity moved in 5 db steps down toward threshold. No subject seemed confused by the instructions, or acted inconsistently except as the region of psychophysical indeterminacy was approached.

After practice and an opportunity to ask any question thresholds were collected at the stated audiometer frequencies 125, 250, 500, 1000, 2000, 4000, and 8000 cps.* At each frequency, upon the first sign that the subject was uncertain whether he heard a tone, the intensity was increased 5 db or until clear-cut responses were obtained. At that point, the intensity was decreased in 1 db steps, allowing at least three spurts per level. Threshold was considered to be that intensity setting last heard two of three times. The intensity was then decreased 5 db and an ascending series in 1 db steps begun. Threshold was considered to be at that setting first heard two of three times. Another descending and anothe ascending series were given, and the final reading noted down as the mean setting of the four threshold-crossings, to the nearest whole number. Of course, human response being what it is, no audiometrician can hold without any deviation to a rigid plan, but very few liberties were taken with this pattern.

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* These frequencies were determined by a Conn Chromatic Stroboscope to be: 125.7, 251.1, 497.9, 988.6, 2046.2, [illegible] and [illegible] cps.

At the conclusion of the seven thresholds, all extraneous attenuation was removed, the audiometer dial was turned to 70 db over zero, and the canal SPL read for each frequency. The audiometer was calibrated to the nearest 0.1 db every day on the 9A coupler; from all this information could be derived the subject's hearing loss with respect to the American closed-coupler standard, and finally the SPL in the ear canal at threshold could be computed.

RESULTS.

The mean and median threshold SPLs, and the standard deviations are given in Table II.

TABLE II.

SOUND PRESSURE LEVEL AT THRESHOLD, MEASURED BY PROBE TUBE UNDER EXPERIMENTAL PHONE, AND TRANSFERRED TO THE PDR-8 PHONE BY LOUDNESS BALANCING.

cps	Median	Mean	Standard Deviation	Probe Tube Attenuation
125	38.5	37.9	7.41	-2.4
250	24.4	24.5	5.46	-7.7
500	18.7	18.9	4.77	4.5
1000	11.3	11.9	5.88	8.2
2000	12.4	14.1	7.35	12.4
4000	15.2	17.5	10.90	19.8
8000	25.1	26.4	16.40	27.4

From the shapes of the distributions, as well as from other considerations, the writer concludes that the median is the more useful measure, and it is the median column in Table II which has been plotted in Fig. 5. This graph is our best estimate to date of canal SPL at threshold for normal-hearing ears in this country. It confirms within two or three db (except at 8000 cps) a previous estimate by the writer* on 20 ears selected in a manner identical to this study, but with a lighter earpho[illegible] and with a somewhat different probe tube.

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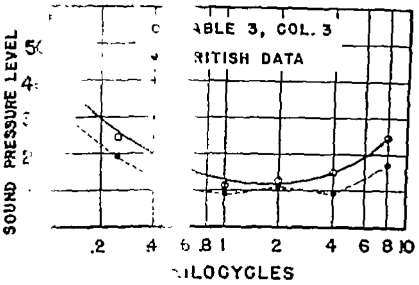
* Unpublished mimeographed memorandum. "Notes on the International Specification of 'Normal Hearing'." U. S. N. Medical Research Laboratory, 1 October, 1953.

In this fi[illegible] [illegible]lson and King's data are replotted for easy compar[illegible] [illegible]tish thresholds are 2-8 db fainter. These differen[illegible] [illegible] explained in one or more of three

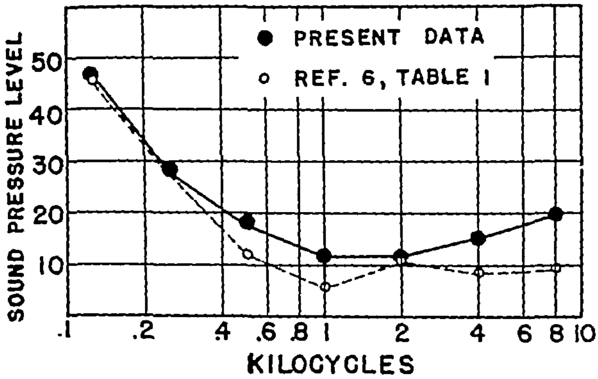


COMPARISON OF PRESENT DATA ON SPL AT THRESHOLD IN EAR CANAL, WITH BRITISH DATA[6]

NOTE: DIFFERENCES MAY BE EQUIPMENTAL

FIG. 5

ways: *1.* Assuming more rigid selection on the part of the British; but this does not seem likely since a somewhat more rigid selection (use of the clinical audiogram) was used for the present data; *2.* The rather amusing assumption that British ears are keener than American, and *3.* Assuming that the difference lies in the two sets of data being dependent upon the specific earphone, earphone-cushion combination, artificial ear or closed coupler, manner of calibrating the probe tube, and perhaps other factors. It seems most likely that here we will find the sources of discrepancy, for a final adjudication. Already a serious discrepancy has been observed (see our Fig. 1) as to just which canal SPL constitutes American "Normal Hearing," as between the 705A phone and the 552 phone, even though the two units had been balanced in loudness and should, one would suppose, yield the same datum. It can well be imagined that if the British 4026A phone used by Dadson and King were loudness-balanced with one of the American standard phones, different ear canal pressures might likewise be developed — enough different to account for the divergences between the data of this paper and that of Dadson and King. Only a full exchange of equipment, with data obtained on identical ears, will of course settle this question.



COMPARISON OF PRESENT DATA ON SPL AT THRESHOLD IN A CLOSED COUPLER, WITH BRITISH DATA[6]

FIG. 6

That equipment factors may lie at the basis of the British-American differences is shown by Fig. 7, in which the two

TABLE III.

PROPOSALS FOR A NEW LABORATORY HEARING STANDARD

	A			B		
	BY PROBE TUBE UNDER PDR-8 PHONE			BY 9A COUPLER AND PDR-8 PHONE		
cps	Present Standard (from Table I, Col. 1)	Median Hearing Loss, Present Group	Proposed Standard, 1 db-Step Procedure	Present Standard From Ref. 15 (-2.5 db)	As in Col. (2)	Proposed Standard, Procedure 1 db-Step
125	44.3	-5.8	38.5	49.9	-5.8	44.1
250	35.0	-10.6	24.4	36.3	-10.6	25.7
500	25.2	-6.5	18.7	22	-6.5	15.5
1000	17.2	-5.9	11.3	14	-5.9	8.1
2000	17.6	-5.2	12.4	14.6	-5.2	9.4
4000	14.3	0.9	16.2	12	0.9	12.9
8000	33.4	-8.3	25.1	25.8	-8.3	17.5

populations are compared on the basis of closed-coupler measurements. At two or three frequencies the differences are negligible, and reach a maximum difference at 8000 cps, where coupler measurements are especially dependent upon design factors.

DISCUSSION.

The smooth curve drawn through our data in Fig. 5 can be accepted as a good estimate of Minimum Audible Pressure at the entrance to the ear canal, providing the phone used is a W.E. 705A or one of the Permoflux series, and providing the probe tube is calibrated in the manner described above. The curve should be useful as a standard of young, healthy ears, to any laboratory or other activity interested in, and equipped to measure, Minimum Audible Pressure in this way.

For activities not equipped with a probe tube but with access to a 9A coupler, the smooth curve in Fig. 5 translates into coupler SPL as shown in Table III, Col. 6. For activities with well-calibrated audiometers, but with no direct access to a 9A coupler, it is only necessary to correct the "zero," on the audiometer dial, by the amount shown in Col. 5, Table III, "Hearing Loss." This new zero then is a fair standard to expect a group of young, healthy ears to meet; however, the usefulness in general clinical audiometry of the smooth curve in Fig. 5 rests upon two subsidiary questions: *1.* A decision as to what sort of population audiometric zero should be based upon, and *2.* What allowance should be made, in setting audiometric zero, for the fact that many, if not most, audiograms are collected under conditions far from ideal.

1. What population should audiometric zero be based on?

It seems clear that a basic population should be unselected except that known hearing defects should be eliminated. It seems to the writer that there is no especial reason why one should not accept the definition of the 1242 persons in the Public Health survey as unselected, if one wishes to obtain an average for a very wide range of age, exposure to noise medical history, intelligence, anxiety in a clinic, and a whole host of other factors which might influence threshold data. The only defect is that individuals of advanced age, with a certainty of some presbyacusis, were included. Unfortunately, the Public Health report never gave the mean threshold for those persons aged 20-29 who formed their fraction of the total 1242 persons on which American "Normal Hearing" rests. If one had this figure, the writer would look no further for an unselected population upon which to base audiometric zero.

A misconception has arisen with respect to the 684 males, aged 20-29, with normal hearing *for speech*, the mean thresholds for which were given by the Public Health report. These 684 men were drawn, not from the 1242 individuals on whom American "Normal Hearing" rests, but from the much larger group of 4662 persons with normal hearing *for speech*, but not necessarily with no audiometric variation greater than 20 db. Of this 4662 group, only 1242, or about 26 per cent, could meet this additional audiometric requirement, and the hearing of the 684 young men was actually worse by 5-6 db at 4096 and 8192 cps, than that of 1242 persons of all ages.

It seems evident, then, that it is not proper to use this group, with known hearing losses, in discussing audiometric zero; nevertheless, it is clear that using only data from the age range 20-29 does not in large surveys improve mean acuity by very much.

Fig. 7 of our previous reference (see footnote*) compares three large surveys in this respect, where the zero reference is the threshold SPL for the ages 20-29, and the curves are the same datum for the age range 10-59 or even more. For the Public Health and San Diego County Fair surveys, differences between the two age ranges are very small, never exceeding 3 db. For the World's Fair survey, on the other hand, differences reach 6-7 db for high tones; but this is perhaps explained by the fact that in that survey the number of individuals used to represent each decade of life was artificially kept approximately constant; there being, for example, more cases at the decade 40-49 than for the decade 10-19. This results in weighting unduly the high-tone losses to be expected in the older groups. Unfortunately, the age distribution of the group of laboratory observers" . . . "For good ears under favorable conditions, the hearing threshold is represented by the MAP curve; on the other hand, for those in the population who report no difficulty in hearing, the threshold as obtained with

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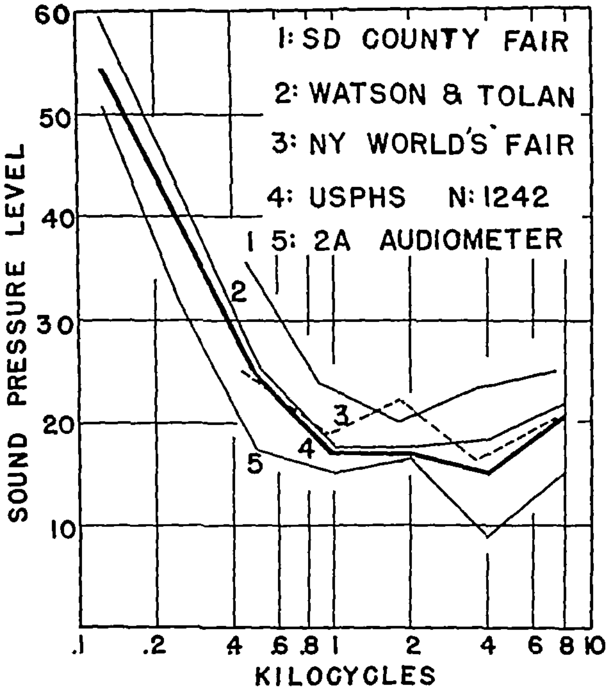
* Unpublished mimeographed memorandum. "Notes on the International Specification of 'Normal Hearing'." U. S. N. Medical Research Laboratory, 1 October, 1953.



AMERICAN "NORMAL", IN 9A COUPLER, COMPARED WITH FOUR OTHER SURVEYS
FIG. 7

an audiometer in the hands of the otologist is probably better represented by the curves corresponding to the zero setting of the audiometers" (p. 277).

To prove their point, Steinberg and Gardner compared the data of Sivian and White, taken under the best conditions at 1000 cps, with data from 100 inexperienced observers;[21] the

Sivian and White threshold was about 7 db fainter (*writer's* note: actually, 10 db fainter than Munson's group). This difference was ascribed to care in training and testing the subjects.

The *correction* of 7 db for practice, rapport, etc., does not wholly explain the discrepancies of up to 12 db, as seen in Fig. 4, between Munson's data and the American standard; moreover, it is likely that the correction is too large. Davis, *et al.* published average "hearing loss" at 500, 1000, and 2000 cps [illegible] 21 partially deafened ears by two procedures, a clinical [illegible]ogram and a laboratory audiogram in 0.5 db steps. He[illegible] loss was found to be 57 db by clinical examination, but [illegible] laboratory test. The difference of 8 db in favor of the [illegible]ratory test is sometimes cited as corroborating the im[illegible]nce of the practice and rapport factors; but a closer loo[illegible] the data causes the "factors" to vanish altogether. "H[illegible] Loss" was not referred to the same SPL in the two cas[illegible] the case of the laboratory test, loss was with refere[illegible] Sivian and White's MAP, which, averaged at the th[illegible]equencies used, equals 9 db re reference pressure. The he[illegible]reshold of these patients by laboratory test, then, was [illegible]dy 9 + 65 ± 74 db. Now the average SPL at thes[illegible] frequencies by American standards (upon which the c[illegible]xamination rests) is 19.5 db. The hearing threshold by clinical test was, then, 19.5 + 57 — 76.5 db, a matter of only 2.5 worse than the 74 db by laboratory test. And even this discrepancy vanishes if one corrects the clinical audiogram for the coarseness of the 5-db step attenuator.

The almost identical clinical and laboratory audiograms of an earlier paper from this Laboratory* have already been cited. Evidently, without too elaborate precautions, clinical audiograms need not differ markedly from more careful appraisal.

In summing up the evidence on a training factor, the writer concludes that a reasonably careful clinical examination need not appreciably suffer from the subjects' being unfamiliar with audiometry, and that no correction need be added *for*

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* Unpublished mimeograph memorandum, "Notes on the International Specification of 'Normal Hearing.' U. S. N. Medical Research Laboratory, 1 October, 1951.

that reason to the laboratory standards of Table III in order to use them for general audiometry. It is true that the evidence upon which this conclusion rests comes from populations of at least average intelligence (*i.e.*, the clinical patients of Davis, *et al.*, the subjects of the writer's previous study, and the subjects of Wheeler and Dickson); nevertheless any correction for very low intelligence should be made on an individual basis and should not influence the standard itself.

Although training as such can be practically disregarded, there remain many other factors which do actually affect clinical audiograms adversely. Should the standard be adjusted to allow for the irregularities and imprecisions which exist in many clinical situations? The writer feels that the audiometer should be adjusted to the best median performance of young, healthy ears, and that it is unjustified to relax the intensity standard to take account of faulty practice. If there are audiometric workspaces in which no threshold closer than within 15 db of "Normal" can be obtained, or lax audiologists, or audiometers routinely out of calibration, there is no necessity to relax the intensity standards so that those being tested under such conditions are not penalized in some way. It seems more pertinent to seek by education, etc., to close the clinical-laboratory gap rather than to accept a large differential as a necessary evil.

It is true, of course, that the present American standard correctly describes the hearing of large populations. Fig. 7 compares the standard in terms of SPL in 6 cc. couplers with four of the more carefully-done surveys, namely, the original calibrating group for the 2A audiometer, the New York World's Fair group, the San Diego County Fair group, and a group of 100 young adult ears reported by Watson and Tolan. The entries in Fig. 7 were derived as follows:

(*a*) *Public Health Survey.* SPL in the 9A coupler is given directly[2,3] for the 1242 "Normal-hearing" persons.

(*b*) *Original calibrating group for the 2A audiometer.* We know[14] the difference between the 2A "Normal" and the American standard; subtracting this difference gives the 9A coupler SPL for the 2A group as follows:

125	250	500	1000	2000	4000	8000	
54.5	39.6	21.8	16.7	17.0	15.1	20.9	American Standard, 705A phone
3.7	8.0	7.3	1.5	0.6	6.1	5.7	PHS correction to the 2A coupler SPL inferred.
50.8	31.6	17.5	15.2	16.4	9.0	15.2	

(c) *New York World's Fair Group.* One can calculate from (*b*) above, by interpolation, the coupler SPL at "Normal" for the 2A calibrating group at the five frequencies used at the World's Fair; Steinberg, *et al.*[23] present a graph showing the db difference between this datum and the reference group (men and women, age 20-29) upon which the World's Fair "Hearing Loss" was based. Subtracting this difference from the coupler SPL for the 2A group, then, gives the coupler SPL for the World's Fair "Normal." Hearing loss of the whole population of the World's Fair group can then be calculated in coupler SPL. This process is shown as follows:

	cps				
	440	880	1760	3520	7040
(1)	20	15.5	16	10	13.5
(2)	+ 0.5	0.0	+ 2.5	+ 0.5	0.0
(3)	20.5	15.5	18.5	10.5	13.5
(4)	+ 2.9	+ 2.9	+ 3.5	+ 6.2	+ 7.1
(5)	23.4	18.4	22.0	16.7	20.6

(1) Interpolated coupler SPL for W.E. 2A calibrating group.

(2) Difference between (1) and the World's Fair "Normal" group (men and women, age 20-29).

(3) Inferred coupler SPL for World's Fair "Normal" group.

(4) Difference between (3) and 35,589 selected ears in the World's Fair group, age 10-59, calculated from Table II, p. 293, ref. 22.

(5) Inferred coupler SPL for total World's Fair group.

(*d*) *The San Diego County Fair Group.* A total of 3482 people aged 10-59, 57 per cent men, were tested binaurally at the same frequencies as at the World's Fair.[24] (A different coupler and a different earphone-cushion combination were used than in the other studies in Fig. 7.) The mean SPLs at threshold for the stated "Normal" group (all 788 men and women aged 20-29) were as follows, compared with the mean differences (*i.e.*, "Hearing Loss") for the whole group of 3482 individuals:

	440	880	1760	3520	7040
(1)	34	22	18	21	22
(2)	1.1	1.3	2.0	2.5	2.8
(3)	35.1	23.3	20.0	23.5	24.8

(1) Mean threshold SPL in 6 c.c. coupler for "Normal" group.

(2) Difference between (1) and threshold for all 3482 people (calculated from their Table III, p. 481).

(3) Coupler SPL at threshold for all participants aged 10-59.

(*e*) *Watson and Tolan's Group.* These 100 young adult ears were very carefully examined with a commercial audiometer in a soundproof room. No history of hearing defect was included, or any subject with any loss of more than 15 db at any frequency. Subjects were naive. This group, then, was selected by very much the same criteria as the group chosen by the Public Health survey to represent "Normal" except that Watson and Tolan's subjects were all young. They reported mean threshold in terms of the American standard, from which the threshold coupler SPL can easily be computed.

A really detailed comparison of these surveys would be out of place here, but a comment must be made bearing on the question, how well is the present American standard substantiated by other surveys.

It is clear from Fig. 7 that the standard is not too lenient, if one reasons from the fact that large-scale surveys do rather well substantiate it. The World's Fair group approximated the American standard within 1 - 3 db except at 1760 cps, where the Fair population had a *loss* of about 5 db. The San Diego County Fair group yielded mean thresholds with hearing *losses* of 3 - 8 db by American standards, and, of course, the Public Health survey itself, considering all 9324 ears of all ages normal for speech, yielded thresholds which agreed with the American standard within less than 1 db, except at 3520 and 7040 cps, where hearing *losses* of 7 - 9 db occurred.

Had the American standard been too lenient, it would have occurred that the survey data would show not *losses*, but the reverse.

Evidently, then, the American standard is an approximately correct statement of the hearing of large populations unselected for age, previous hearing history, etc., but this is not to say that these large populations, with known hearing defects, should serve to characterize a standard for unimpaired hearing.

In the matter of setting standards, it is necessary to balance one good against another. To keep the present standard is to maintain the rehabilitation load as we know it today; to adopt a standard such as in our Table III would have the effect of earlier identification of defects. A child with a loss of, say, 5 db at 500 cps is now thought to be perfectly normal within limits of experimental error, but upon adopting the standards in Table III, that child would seem in reality to have a loss of more than 10 db, and to warrant immediate close observation. If many children exhibited such losses, then the workspace, or the technique, or the equipment would be laid under suspicion; thus a general improvement in audiometry would result.

In the writer's opinion, these two benefits outweigh rather heavily the advantage of retaining the present standard, which has only the effects of condoning laxity and of deferring (at the expense of the patient's well-being) the initiation of therapy.

SUMMARY.

The history is briefly reviewed of how American "Normal Hearing" came to be standardized. It was shown that the standard could be specified in terms of sound pressure level in the ear canal as well as in a brass coupler as at present.

The discrepancy usually cited as excessive between hearing norms as collected by large clinical surveys and by laboratory studies is shown to be sharply reduced when the quite correct comparisons are made. Nevertheless, an appreciable discrepancy, though not exceeding 8 db, exists.

It is reasoned that the clinical-laboratory discrepancy is

not due to selection of subjects, nor of familiarity of subjects with psychoacoustic judgments, but resides in features of the clinical situation which are correctable.

A laboratory standard for audiometer "Normal Hearing" is proposed which would more nearly correctly describe the status of an individual's hearing ability in comparison with young, healthy ears, and which would tend at the same time to have a salutary effect upon audiometric practice.

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POST GRADUATE SESSION IN OPHTHALMOLOGY AND OTOLARYNGOLOGY.

A Post Graduate Session in Ophthalmology and Otolaryngology sponsored by the Virginia Society of Ophthalmology and Otolaryngology will meet at the University of Virginia from November 30 to December 3, 1954.

November 30 and December 1, will be devoted to Otolaryngology lectures and December 2 and 3, will be devoted to Ophthalmological subjects. Those interested please contact Dr. Edwin Burton or Dr. G. Slaughter Fitz-Hugh, 104 East Market Street, Charlottesville, Va.

THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC., MEETINGS.

MID-WINTER.

Eastern Section Meeting..............................Friday, January 7, 1955
Philadelphia, Pa., Hotel Warwick

Triological Council Meeting....................Saturday, January 8, 1955
New York City, The Waldorf-Astoria

Western Section Meeting...Saturday-Sunday, Jan. 15-16, 1955
Los Angeles, Cal., The Town House

Southern Section Meeting...................Saturday, January 22, 1955
Charlottesville, Va., Medical School Auditorium

Middle Section Meeting..........................Monday, January 24, 1955
Detroit, Mich., Sheraton-Cadillac Hotel

SPRING.

American Board of Otolaryngology
Richmond, Va., March 6-10, 1955

All Society Meetings will be held at the Hollywood Beach Hotel, Hollywood, Florida.

American Laryngological Association.............March 13, 14, 1955
Triological Society.....(mornings only) March 15, 16, 17, 1955
American Broncho-Esophagological Association
(afternoons only) March 15, 16, 1955
American Otological Society, Inc.......................March 17, 18, 1955

The Hollywood Beach Hotel (American plan) is a delightful place to hold the Spring Meetings — you may wish to bring the family along. The Hotel offers its guests such features as ocean bathing, deep sea fishing, 18-hole golf course and country club, tennis and many other activities.

For hotel reservations please communicate with Mr. John W. Tyler, Manager, Hollywood Beach Hotel, Hollywood, Florida.

TWENTY-FOURTH ANNUAL MID-WINTER CLINICAL CONVENTION IN OPHTHALMOLOGY AND OTOLARYNGOLOGY, JANUARY 17 THROUGH JANUARY 28, 1955.

The Research Study Club of Los Angeles announces its Twenty-fourth Annual Mid-Winter Clinical Convention in Ophthalmology and Otolaryngology, January 17 through January 28, 1955. For the Ear-Nose-Throat, the main Guest Lecturers will include Dr. Theodore E. Walsh, Professor and Head of the Department of Otolaryngology, Washington University, St. Louis, and Dr. John J. Conley, Chief of the Head and Neck Service, St. Vincent's Hospital, New York City, and Clinical Professor of Otorhinolaryngology, New York University, and other appointments.

Dr. Walsh has long been known to us not only as a professor but also as a lecturer in our national societies, with particular interest in otologic and rhinologic problems.

Dr. Conley has done a great deal of work in tumors and plastic surgery of the head and neck, as well as being a well trained man in the broader field of our specialty.

Also serving on our program with Didactic Lectures will be Dr. Kenneth C. Brandenburg of Long Beach, Dr. J. C. Alroy Harding of San Diego, and Dr. Paul J. Moses of San Francisco.

Instruction Courses in Otolaryngology will be provided by Dr. E. R. V. Anderson, Dr. Norman Jesberg, Dr. Harold Owens, and Dr. Manuel R. Wexler, all of Los Angeles, and Dr. Leo Shahinian of San Francisco.

The main Guest Lecturers for the Eye program will be Dr. Edmund B. Spaeth and Dr. Irving H. Leopold, both from the University of Pennsylvania Graduate School of Ophthalmology.

The first week will be devoted to the Ear-Nose-Throat—from Monday to Saturday, January 17 through January 22. The Eye week will cover Monday to Friday, January 24 through January 28. Those of us who confine our work to only one of these specialties may complete either subject in one week.

On Tuesday evening, January 18, the Annual Banquet of the Los Angeles Society of Ophthalmology and Otolaryngology will honor the Guest Lecturers of that week. This will be held at 7:00 p.m. in the Ball Room of the Elks Club.

On Wednesday afternoon of each week motion pictures will be shown, Ear-Nose-Throat subjects the first week, and Eye subjects the second week. These afternoons will also afford time for relaxation, sight-seeing, golf, radio broadcasts, and the enjoyment of points of interest in and about Los Angeles, including the famous Huntington Library with its collection of rare books and art.

The American Laryngological, Rhinological and Otological Society, Western Section, will hold its Annual Meeting at The Town House immediately preceding this Convention, Saturday and Sunday, January 15 and 16. On Saturday afternoon the scientific session is open to members and all interested otolaryngologists. Saturday evening there will be a dinner meeting for members of the Triologic Society only. Sunday morning, the second scientific session is open to all interested. The president of the Society, Dr. Kenneth M. Day, after attending this Society's Annual Meeting, will be with us during a portion of the Mid-Winter Course of the Research Study Club.

Unless you have your own plans for a place to stay, it is advised that you write for reservations at an early date to Mr. H. M. Nickerson, Manager of the Elks Club, 607 South Parkview Street, Los Angeles 57. He will endeavor to arrange suitable quarters for you in a conveniently located hotel, and will confirm the reservation by return mail.

Each applicant must be a member, in good standing, of the American Medical Association *in Order to Become Eligible* for attendance at the Convention. The fee for the entire two weeks, or any part of it, is $100.00 and includes the cost of all luncheons. We are informed that these dues are an income-tax deductible item, as they represent an annual membership fee. Make your Check payable to "Mid-Winter Clinical Convention," and mail to Pierre Violé, M.D., Treasurer, 1930 Wilshire Boulevard, Los Angeles 57. If anything prevents your attendance, this fee will be returned to you.

UNIVERSITY OF FLORIDA MID-WINTER SEMINAR.

The Ninth Annual University of Florida Mid-winter Seminar in Ophthalmology and Otolaryngology will be held at the Sans Souci Hotel in Miami Beach, the week of January 17, 1955. The lectures on Ophthalmology will be presented on January 17, 18, and 19, and those on Otolaryngology on January 20, 21, and 22. A mid-week feature will be the Midwinter Convention of the Florida Society of Ophthalmology and Otolaryngology on Wednesday afternoon, January 19, to which all registrants are invited. The registrants and their wives may also attend the informal banquet at 8 p.m., on Wednesday. The Seminar schedule permits ample time for recreation.

The Seminar lecturers on Ophthalmology this year are: Dr. William F. Hughes, Jr., Chicago; Dr. Phillips Thygeson, San Jose; Dr. James Allen, New Orleans; Dr. Walter H. Fink, Minneapolis; and Dr. Milton L. Berliner, New York. Those lecturing on Otolaryngology are: Dr. Paul Holinger, Chicago; Dr. Lawrence R. Boies, Minneapolis; Dr. Edmund P. Fowler, Jr., New York; Dr. Arthur W. Proetz, St. Louis, and Dr. David DeWeese, Portland, Oregon.

ANNOUNCEMENT.

An introductory course in Reconstructive Surgery of the Septum and External Nasal Pyramid will be given, under the direction of Dr. Maurice Cottle, March 19 through 26, 1955, at the University of Oregon Medical School. This will be sponsored by the Department of Otolaryngology.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

November 1, 1954.

Acousticon Models A-17, A-180, A-185 and A-1530.
Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 1, New York.

Auditone Models 11 and 15.
Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67 and 70.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac Model M; Mono-Pac Model "Lyric"; Mono-Pac Model "Rhapsody."
Manufacturer: Beltone Hearing Aid Co., 2900 West 36th St., Chicago 32, Ill.

Cleartone Model 700.
Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Junior Model D-2; Dahlberg Model D-3 Tru-Sonic; Dahlberg Model D-4 Tru-Sonic.
Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Fortiphone Models 19-LR; 20A; 21-C and 22.
Manufacturer: Fortiphone Limited, Fortiphone House, 247 Regent St., London W. 1, England.
Distributor: Anton Heilman, 75 Madison Ave., New York 16, N. Y.

Gem Hearing Aid Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.
Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.
Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico Top Secret Model L; Maico Maxitone.
Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis, Minn.

Micronic Model "Mercury."
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Model T-10; Microtone Model T-612.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

Normatone Model C and Model D-53.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.

Distributor: Normatone Hearing Aid Co., 23 East 7th St., St. Paul (1), Minn.

Otarion Models B-15 and B-30; Otarion Models F-1, and F-3; Otarion Model H-1; Custom "5."

Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40, Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny Myte).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Model 62 Starlet; Model 72; Model 82 (Zephyr).

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Distributor: Radioear Corp., 306 Beverly Ro., Mt. Lebanon, Pittsburgh 16, Pa.

Silvertone Model H-16, J-92; Silvertone Model P-15.

Manufacturer: W. E. Johnson Mfg. Co., 703 W. 40th St., Minneapolis, Minn.

Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966; Sonotone Model 977; Sonotone Model 988.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Televox Model E.

Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 99; Telex Model 200; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 953; Telex Model 1700.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.

Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster; Model Cameo.

Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill

Unex Models 200 and 230.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal; Zenith "Regent."

Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices have vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TRANSISTOR HE ARING AIDS ACCEPTED.

Acousticon Model A300; 1 transistor, 2 tubes. Model A-310; 1 transistor; 2 tubes. Model A-330; 3 transistors. Model A-335; 3 transistors.

Manufacturer. Dictograph Products, Inc., 95-25 149th St., Jamaica 35, New York.

Audivox, Model 71; 3 transistors.

Manufacturer: Audivox, Inc., 123 Worcester St., Boston 18, Mass.

Beltone Concerto Model; 3 transistors.

Manufacturer: Beltone Hearing Aid Co., 2900 W. 36th St., Chicago 32. Illinois.

Maico Transist-Ear, Model O; 3 transistors.

Manufacturer: The Maico Company, Inc., 21 N. 3rd St., Minneapolis 1. Minnesota.

Micronic "All American" Hearing Aid; 3 transistors.

Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Model T1 (Red Dot); 3 transistors. Microtone Model T1 (Yellow Dot); 3 transistors. Microtone Model T31 (Micro-Mite); 1 transistor and 2 tubes. Microtone Model Skylark; 3 transistors.

Manufacturer: The Microtone Corporation, Ford Parkway on the Mississippi, St. Paul 1, Minn.

Otarion Model C-15; 1 transistor, 2 tubes. Otarion Model D-1; 3 transistors. Otarion Model F-22; 1 transistor, 2 tubes.

Manufacturer: Otarion, Inc., 4757 N. Ravenswood Ave., Chicago 40, Ill

Paravox Model K.M. (TRANSonic); 3 transistors.

Manufacturer: Paravox, Inc., 2056 East 54th St., Cleveland, Ohio.

Radioear Model 820; 3 transistors.

Manufacturer: E. A. Myers & Sons, Inc., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Sonotone Model 1010; 1 transistor, 2 tubes. Model No. 1111; 3 transistors.

Manufacturer: Sonotone Corporation, Elmsford, N. Y.

Telex Model 954; 1 transistor, 2 tubes. Telex Model 956; 3 transistors.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Unex Model TR-3D; 3 transistors.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Zenith Model Royal-T; 3 transistors. Zenith Model Super Royal-T; 3 transistors.

Manufacturer: Zenith Radio Corp., 5801 W. Dickens Ave., Chicago 39, Illinois.

SEMI PORTABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).

Manufacturer: A. M. Brooks Co., 1222 W. Washington Blvd., Los Angeles 7, Calif.

Aurex Hearing Aids (three types).

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.

Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. D. E. Staunton Wishart, 170 St. George St., Toronto 5, Ontario, Canada.
Vice-President: Dr. Wm. J. McNally, 1509 Sherbrooke St., West Montreal 25, Canada.
Secretary-Treasurer: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Illinois.
Editor-Librarian: Dr. *Henry L. Williams*, Mayo Clinic, Rochester, Minn.
Meeting: Hollywood Hotel, Hollywood, Fla., March 17 (afternoon), 18 (forenoon and afternoon), 19 (forenoon), 1955.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. Henry B. Orton, Newark, N. J.
First Vice-President: Dr. Jas. H. Maxwell, Ann Arbor, Mich.
Second-Vice-President: Dr. Clyde A. Heatley, Rochester, N. Y.
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Treasurer: Fred W. Dixon, Cleveland, Ohio.
Librarian-Historian: Dr. Bernard J. McMahon, St. Louis, Mo.
Meeting: Hollywood Hotel, Hollywood, Fla., March 13-14, 1955.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Kenneth M. Day, 121 University Pl., Pittsburgh, Pa.
President-Elect. Dr. Dean M. Lierle, Iowa City, Iowa.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Eastern Section Meeting, Friday, January 7, 1955, Philadelphia, Pa., Hotel Warwick.
Triological Council Meeting, Saturday, January 8, 1955, New York City, The Waldorf-Astoria.
Western Section Meeting, Saturday - Sunday, January 15 - 16, 1955, Los Angeles, Cal., The Town House.
Southern Section Meeting, Saturday, January 22, 1955, Charlottesville, Va., Medical School Auditorium.
Middle Section Meeting, Monday, January 24, 1955, Detroit, Mich., Sheraton-Cadillac Hotel.
Meeting: Hollywood Hotel, Hollywood, Fla., March 15-16-17, 1955, morning only.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. F. W. Davison, Danville, Pa.
Vice-Chairman: Dr. Guy L. Boyden, Portland, Ore.
Secretary: Dr. Hugh A. Kuhn, Hammond, Ind.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Algernon B. Reese, 73 East 71st St., New York 21, N. Y.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., October 9-15 [illegible]955.

AMERICAN BOARD OF OTOLA[illegible]Y.

Meeting: Hotel [illegible] Richmond, Va., M[illegible].
Pa[illegible] Chicago, Ill., Octo[illegible]

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Clyde A. Heatley, 11 No. Goodman St., Rochester, N. Y.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Square, Philadelphia, Pa.
Meeting: Hollywood Beach Hotel, Hollywood, Fla., March 15-16, 1955, afternoon only.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. John Louzan.
Vice-Chairman: Dr. Willard B. Walters.
Secretary: Dr. Jack L. Levine.
Treasurer: *Dr. Russell S. Page, Jr.*
Meetings are held on the third Tuesday of October, November, March and May, 7:00 P.M.
Place: Army and Navy Club, Washington, D. C.

GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Meeting: General Oglethorpe Hotel, Savannah, Ga., *March 11-12, 1955.*

THE GREATER *MIAMI* EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. George McKenzie, Dupont Medical Bldg., Miami, Fla.
Vice-President: Dr. Maurice Edelman, 605 Lincoln Road, Miami Beach, Florida.
Secretary: Dr. James H. Mendel, Jr., 7241 Red Road, Miami, Fla.
Meeting: Quarterly, at Seven Seas Restaurant, February, May, October, and December.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

President: Dr. Norman Leshin, 55 East Washington St., Chicago, Ill.
Vice-President: Dr. Henry B. Perlman, 950 East 59th St., Chicago, Ill.
Secretary-Treasurer: Dr. Stanton A. Friedberg, 122 So. Michigan Ave., Chicago, Ill.
Meeting: First Monday of each Month, October through May.

THE LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. W. L. Hughes, Lamar Life Bldg., Jackson, Miss.
Vice-President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

OTOSCLEROSIS STUDY GROUP.

President: Dr. Gordon D. Hoople, 1100 East Genesee St., Syracuse, N. Y.
Secretary: Dr. Lawrence R. Boies, Med. Arts Bldg., Minneapolis 2, Minn.
Meeting: Palmer House, Chicago, Ill., Oct. 9, 1955.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. F. Lambert McGannon, 14900 Detroit Ave., Lakewood 9, Ohio.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2, N. Y.
Meeting: Palmer House, Chicago, Ill., October, 1955.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this *information* up to date).

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. D. E. Staunton Wishart, 170 St. George St., Toronto 5, Ontario, Canada.
Vice-President: Dr. Wm. J. McNally, 1509 Sherbrooke St., West Montreal 25, Canada.
Secretary-Treasurer: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Illinois.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Hollywood Hotel, Hollywood, Fla., March 17 (afternoon), 18 (forenoon and afternoon), 19 (forenoon), 1955.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. Henry B. Orton, Newark, N. J.
First Vice-President: Dr. Jas. H. Maxwell, Ann Arbor, Mich.
Second-Vice-President: Dr. Clyde A. Heatley, Rochester, N. Y.
Secretary: Harry P. Schenck, Philadelphia, Pa.
Treasurer: Fred W. Dixon, Cleveland, Ohio.
Librarian-Historian: Dr. Bernard J. McMahon, St. Louis, Mo.
Meeting: Hollywood Hotel, Hollywood, Fla., March 13-14, 1955.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Kenneth M. Day, 121 University Pl., Pittsburgh, Pa.
President-Elect. Dr. Dean M. Lierle, Iowa City, Iowa.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Eastern Section Meeting, Friday, January 7, 1955, Philadelphia, Pa., Hotel Warwick.
Triological Council Meeting, Saturday, January 8, 1955, New York City, The Waldorf-Astoria.
Western Section Meeting, Saturday - Sunday, January 15 - 16, 1955, Los Angeles, Cal., The Town House.
Southern Section Meeting, Saturday, January 22, 1955, Charlottesville, Va., Medical School Auditorium.
Middle Section Meeting, Monday, January 24, 1955, Detroit, Mich., Sheraton-Cadillac Hotel.
Meeting: Hollywood Hotel, Hollywood, Fla., March 15-16-17, 1955, morning only.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. F. W. Davison, Danville, Pa.
Vice-Chairman: Dr. Guy L. Boyden, Portland, Ore.
Secretary: Dr. Hugh A. Kuhn, Hammond, Ind.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Algernon B. Reese, 73 East 71st St., New York 21, N. Y.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., October 9-15, 1955.

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Hotel Marshall, Richmond, Va., March 6-10, 1955.
Palmer House, Chicago, Ill., October, 1955.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Clyde A. Heatley, 11 No. Goodman St., Rochester, N. Y.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Square, Philadelphia, Pa.
Meeting: Hollywood Beach Hotel, Hollywood, Fla., March 15-16, 1955, *afternoon only.*

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. John Louzan.
Vice-Chairman: Dr. Willard B. Walters.
Secretary: Dr. Jack L. Levine.
Treasurer: Dr. Russell S. Page, Jr.
Meetings are held on the third Tuesday of October, November, March and May, 7:00 P.M.
Place: Army and Navy Club, Washington, D. C.

GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Meeting: General Oglethorpe Hotel, Savannah, Ga., March 11-12, 1955.

THE GREATER MIAMI EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. George McKenzie, *Dupont Medical Bldg., Miami, Fla.*
Vice-President: Dr. Maurice Edelman, 605 Lincoln Road, Miami Beach, Florida.
Secretary: Dr. James H. Mendel, Jr., 7241 Red Road, Miami, Fla.
Meeting: Quarterly, at Seven Seas Restaurant, February, May, October, and December.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

President: Dr. Norman Leshin, 55 East Washington St., Chicago, Ill.
Vice-President: Dr. Henry B. Perlman, 950 East 59th St., Chicago, Ill.
Secretary-Treasurer: Dr. Stanton A. Friedberg, 122 So. Michigan Ave., Chicago, Ill.
Meeting: First Monday of each Month, October through May.

THE LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. W. L. Hughes, Lamar Life Bldg., Jackson, Miss.
Vice-President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

OTOSCLEROSIS STUDY GROUP.

President: Dr. Gordon D. Hoople, 1100 East Genesee St., Syracuse, N. Y.
Secretary: Dr. Lawrence R. Boies, Med. Arts Bldg., Minneapolis 2, Minn.
Meeting: Palmer House, Chicago, Ill., Oct. 9, 1955.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. F. Lambert McGannon, *14900* Detroit Ave., Lakewood 9, Ohio.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2, N. Y.
Meeting: Palmer House, Chicago, Ill., October, 1955.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. J. M. Tato, Azcuenaga 235, Buenos Aires, Argentina.
Executive Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U. S. A.
Meeting: Fifth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
President: Dr. J. H. Font, Medical Arts Bldg., San Juan, P. R.
Time and Place: 1956, Puerto Rico.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.
Meeting:

THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. S. Fitz-Hugh, Charlottesville, Va.
President-Elect: Dr. H. L. Mitchell, Lexington, Va.
Vice-President: Dr. Marion K. Humphries, Charlottesville, Va.
Secretary-Treasurer: Dr. L. B. Sheppard, 301 Medical Arts Bldg., Richmond, Va.
Spring Meeting: Natural Bridge Hotel, Natural Bridge, Va., May 7-8, 1955.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President:Harold Owens, M.D.
Secretary-Treasurer: Robert A. Norene, M.D.
Chairman of Section on Ophthalmology: Sol Rome, M.D.
Secretary of Section on Ophthalmology: Wendell C. Irvine, M.D.
Chairman of Section on Otolaryngology: Max E. Pohlman, M.D.
Secretary of Section on Otolaryngology: Herschel H. Burston, M.D.
Place: Los Angeles County Medical Association Building, 1925 Wilshire Boulevard, Los Angeles 57, Calif.
Time: 6:00 P.M., first Thursday of each month from September to June inclusive—Ophthalmology Section. 6:00 P.M., fourth Monday of each month from September to June inclusive—Otolaryngology Section.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Harry Nievert, 555 Park Ave., New York (21), N. Y.
Secretary: Dr. Louis Joel Fleit, 66 Park Ave., New York (16), N. Y.
Meeting:

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. Cecil Swann, Asheville, N. C.
Secretary and Treasurer: Dr. Geo. B. Ferguson, Durham, N. Car.
Meeting: Joint, with South Carolina Society of Ophthalmology and Otolaryngology, Durham, N. C., Nov. 4-6, 1954.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. David S. Asbill, Columbia, S. Car.
Vice-President: Dr. John McLean, Greenville, S. Car.
Secretary-Treasurer: Dr. Roderick Macdonald, Rock Hill, S. Car.
Meeting: Joint, with North Carolina Eye, Ear, Nose and Throat Society, Durham, N. C., Nov. 4-6, 1954.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.
Secretary-Treasurer: Dr. John F. Tolan, 3419 47th Ave., Seattle (5), Wash.
Meeting: Honolulu, 1954.

THE RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Chairman: Dr. Isaac H. Jones, 635 S. Westlake, Los Angeles, Calif.
Treasurer: Dr. Pierre Violé, 1930 Wilshire Blvd., Los Angeles, Calif.
Program Chairmen:
Otolaryngology: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.
Ophthalmology: Dr. Harold F. Whalman, 727 W. 7th St., Los Angeles, Calif.

Mid-Winter Clinical Convention annually the last two weeks in January at Los Angeles, Calif.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Chas. C. Grace, 145 King St., St. Augustine, Fla.
President-Elect: Dr. Jos. W Taylor, 706 Franklin St., Tampa, Fla.
Secretary-Treasurer: Dr. Carl S. McLemore, 1217 Kuhl Ave., Orlando, Fla.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President: Dr. William J. Hitschler.
Vice-President: Dr. Chevalier L. Jackson.
Treasurer: Dr. John J. O'Keefe.
Secretary: Dr. Joseph P. Atkins.
Historian: Dr. Herman B. Cohen.
Executive Committee: Dr. Thomas F. Furlong, Jr., Dr. William A. Lell, Dr. Harry P. Schenck, Dr. Benjamin H. Shuster, ex-officio.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Vice-Chairman: Dr. K. W. Cosgrove, 111 E. Capitol Ave., Little Rock, Ark.
Secretary: Dr. F. A. Holden, Medical Arts Bldg., Baltimore, Md.
Meeting:

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James K. Stewart, Wheeling, W. Va.
Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. C. Otrich, Belleville, Ill.
President-Elect: Dr. Phil R. McGrath, Peoria, Ill.
Secretary-Treasurer: Dr. Alfred G. Schultz, Jacksonville, Ill.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Oscar Marchman, Jr., Dallas, Texas.
Secretary-Treasurer: Dr. Morris F. Waldman, Dallas, Texas.

BALTIMORE NOSE AND THROAT SOCIETY

Chairman: Dr. Albert Steiner, 1308 Eutaw Pl., Baltimore, Md.
Secretary-Treasurer: Dr. Walter E. Loch, 1039 No. Calvert St., Baltimore, Maryland.

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDICO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Salvador Mixco Pinto.
Secretary: Dr. Daniel Alfredo Alfaro.
Treasurer: Dr. Antonio Pineda M.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. J. M. Tato, Azcuenaga 235, Buenos Aires, Argentina.
Executive Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U. S. A.
Meeting: Fifth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
President: Dr. J. H. Font, Medical Arts Bldg., San Juan, P. R.
Time and Place: 1956, Puerto Rico.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.
Meeting:

THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. S. Fitz-Hugh, Charlottesville, Va.
President-Elect: Dr. H. L. Mitchell, Lexington, Va.
Vice-President: Dr. Marion K. Humphries, Charlottesville, Va.
Secretary-Treasurer: Dr. L. B. Sheppard, 301 Medical Arts Bldg., Richmond, Va.
Spring Meeting: Natural Bridge Hotel, Natural Bridge, Va., May 7-8, 1955.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President:Harold Owens, M.D.
Secretary-Treasurer: Robert A. Norene, M.D.
Chairman of Section on Ophthalmology: Sol Rome, M.D.
Secretary of Section on Ophthalmology: Wendell C. Irvine, M.D.
Chairman of Section on Otolaryngology: Max E. Pohlman, M.D.
Secretary of Section on Otolaryngology: Herschel H. Burston, M.D.
Place: Los Angeles County Medical Association Building, 1925 Wilshire Boulevard, Los Angeles 57, Calif.
Time: 6:00 P.M., first Thursday of each month from September to June inclusive—Ophthalmology Section. 6:00 P.M., fourth Monday of each month from September to June inclusive—Otolaryngology Section.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Harry Nievert, 555 Park Ave., New York (21), N. Y.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York (16), N. Y.
Meeting:

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. Cecil Swann, Asheville, N. C.
Secretary and Treasurer: Dr. Geo. B. Ferguson, Durham, N. Car.
Meeting: Joint, with South Carolina Society of Ophthalmology and Otolaryngology, Durham, N. C., Nov. 4-6, 1954.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. David S. Asbill, Columbia, S. Car.
Vice-President: Dr. John McLean, Greenville, S. Car.
Secretary-Treasurer: Dr. Roderick Macdonald, Rock Hill, S. Car.
Meeting: Joint, with North Carolina Eye, Ear, Nose and Throat Society, Durham, N. C., Nov. 4-6, 1954.

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Secretary-Treasurer: Dr. John F. Tolan, 3419 47th Ave., Seattle (5), Wash.
Meeting: Honolulu, 1954.

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Treasurer: Dr. Pierre Violé, 1930 Wilshire Blvd., Los Angeles, Calif.
Program Chairmen:
- Otolaryngology: Dr. Leland G. Hunnicutt, 93 N. Madison Ave., Pasadena, Calif.
- Ophthalmology: Dr. Harold F. Whalman, 727 W. 7th St., Los Angeles, Calif.

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President: Dr. Chas. C. Grace, 145 King St., St. Augustine, Fla.
President-Elect: Dr. Jos. W. Taylor, 706 Franklin St., Tampa, Fla.
Secretary-Treasurer: Dr. Carl S. McLemore, 1217 Kuhl Ave., Orlando, Fla.

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Vice-President: Dr. Chevalier L. Jackson.
Treasurer: Dr. John J. O'Keefe.
Secretary: Dr. Joseph P. Atkins.
Historian: Dr. Herman B. Cohen.
Executive Committee: Dr. Thomas F. Furlong, Jr., Dr. William A. Lell, Dr. Harry P. Schenck, Dr. Benjamin H. Shuster, ex-officio.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Vice-Chairman: Dr. K. W. Cosgrove, 111 E. Capitol Ave., Little Rock, Ark.
Secretary: Dr. F. A. Holden, Medical Arts Bldg., Baltimore, Md.
Meeting:

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James K. Stewart, Wheeling, W. Va.
Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. C. Otrich, Belleville, Ill.
President-Elect: Dr. Phil R. McGrath, Peoria, Ill.
Secretary-Treasurer: Dr. Alfred G. Schultz, Jacksonville, Ill.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Oscar Marchman, Jr., Dallas, Texas.
Secretary-Treasurer: Dr. Morris F. Waldman, Dallas, Texas.

BALTIMORE NOSE AND THROAT SOCIETY

Chairman: Dr. Albert Steiner, 1308 Eutaw Pl., Baltimore, Md.
Secretary-Treasurer: Dr. Walter E. Loch, 1039 No. Calvert St., Baltimore, Maryland.

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDICO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Salvador Mixco Pinto.
Secretary: Dr. Daniel Alfredo Alfaro.
Treasurer: Dr. Antonio Pineda M.

CANADIAN OTOLARYNGOLOGICAL SOCIETY
SOCIETE CANADIENNE D'OTOLARYNGOLOGIE

President: Dr. G. Edward Tremble, 1390 Sherbrooke St., West, Montreal, P. Q.
Secretary: Dr. G. Arnold Henry, 170 St. George St., Toronto, Ontario.
Meeting: Bigwin Inn, Lake of Bays, Muskoka, Ontario. June 16 to June 18, 1955.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

President: Dr. Cesar LaBoide, Mexico, D. F.
Vice-President: Dr. M. Gonzalez Ulloa, Mexico, D. F.
Secretary: Dr. Juan de Dios Peza, Mexico, D. F.

FEDERACION ARGENTINA, DE SOCIEDADES DE OTORRINOLARINGOLOGIA.

Secretario del Exterior: Dr. Juan Manuel Tato.
Sub-Secretario del Exterior: Dr. Oreste E. Bergaglio.
Secretario del Interior: Dr. Eduardo Casterán.
Sub-Secretario del Interior: Dr.Atilio Viale del Carril.
Secretario Tesorero: Dr. Vicente Carri.
Sub-Secretario Tesorero: Dr. José D. Suberviola.

ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

Presidente: Dr. J. Abello.
Vice-Presdente: Dr. Luis Suñe Medan.
Secretario: Dr. Jorge Perelló, 319 Provenza, Barcelona.
Vice-Secretario: Dr. A. Pinart.
Vocal: Dr. J. M. Ferrando.

SOCIEDAD NACIONAL DE CIRUGIA OF CUBA.

Presidente: Dr. Reinaldo de Villiers.
Vicepresidente: Dr. César Cabrera Calderin.
Secretario: Dr. José Xirau.
Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Andre Soulas, Paris, France.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa. U. S. A.
Meeting: 3rd International Congress of Broncho-Esophagology.
Time and Place: September or October, 1954, Lisbon, Portugal.

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER — CAMPINAS.

President: Dr. Heitor Nascimento.
First Secretary: Dr. Roberto Barbosa.
Second Secretary: Dr. Roberto Franco do Amaral.
Librarian-Treasurer: Dr. Leoncio de Souza Queiroz.
Editors for the Archives of the Society: Dr. Guedes de Melo Filho. Dr. Penido Burnier and Dr. Gabriel Porto.

SOCIEDAD CUBANA DE OTO-LARINGOLOGIA.

President: Dr. Reinaldo de Villiers.
Vice-President: Dr. Jorge de Cárdenas.
Secretary: Dr. Pablo Hernandez.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA.

Presidente: Dr. Aldo Remorino.
Vice-Presidente: Dr. Luis E. Olsen.
Secretario: Dr. Eugenio Romero Diaz.
Tesorero: Dr. Juan Manuel Pradales.
Vocales: Dr. Osvaldo Suárez, Dr. Nondier Asís R., Dr. Jorge Bergallo Yofre.

BUENOS AIRES CLUB OTORINOLARINGOLOGICO.

Presidente: Dr. K. Segre.
Vice-Presidente: Dr. A. P. Belou.
Secretario: Dr. S. A. Aranz.
Pro-Secretario: Dr J. M. Tato.
Tesorero: Dr. F. Games.
Pro-Tesorero: Dr. J A Bello.

SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLOMBIA).

Presidente: Dr. Alfonso Tribin P.
Secretario: Dr. Felix [illegible] Lozano.
Tesorero: Dr. Mario Arenas A.

SOCIEDAD ESPANOLA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. D. Adolfo Hinojar Pons.
Vice-Presidente: Dr. D Jose Perez Mateos.
Secretario General Dr. D Francisco Marañés.
Tesorero: Dr. D Ernesto Alonso Ferrer.

ASOCIACION DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DE GUATEMALA

Presidente: Dr. Julio Quevedo, 15 Calle Oriente No. 5.
First Vice-Presidente: Dr. Héctor Cruz, 3a Avenida Sur No. 72.
Second Vice-Presidente: Dr. José Luis Escamilla, 5a Calle Poniente No. 48.
Secretario-Tesorero: Dr. Horace Polanco, 13 Calle Poniente No. 9-D.

FIRST CENTRAL AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY.

President: Dr. Victor M. Noubleau, San Salvador.
Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

SOCIEDAD DE ESTUDIOS CLINICOS DE LA HABANA

President: Dr. Frank Canosa Lorenzo.
Vice-Presidente: Dr. Julio Sanguily.
Secretario: Dr. Juan Portuondo de Castro.
Tesorero: Dr. Luis Ortega Verdes.

FOURTH LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Dario.
Secretary:
Meeting: Lima, Peru, 1957.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY

President: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis, Mo.
General Secretary: Dr. Paul Hollinger, 700 No. Michigan Ave., Chicago (11), Ill.
Meeting: Statler Hotel, Washington, D. C., May 5-10, 1957.

SOCIEDADE PORTUGUESA DE OTORRINOLARINGOLOGIA E DE BRONCO-ESOFAGOLOGIA

Presidente: Dr. Alberto Luis de Mendonca.
Vice-Presidente: Dr. Jaime de Magalhaes.
1.° Secretario: Dr. Antonio da Costa Quinta.
2.° Secretario: Dr. Albano Coelho.
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NUTRITION AND RELATED PROBLEMS.*

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In this thirteenth editorial concerning Nutrition, we present our gleanings from the literature on the five kindred subjects, Nutrition, Endocrines, Autonomic Influence, Stress and Allergy. Although well aware that we are not familiar enough with them, we have come to realize their immediate importance in our daily practice, particularly since otolaryngology has come closer to the field of general medicine.

Nobody can study an article on any one of these five problems without being struck by its close relation to one or more of the others. We have felt the apparent fitness of listing the several contributions under these five headings, yet we particularly emphasize the close relationship which forbids their separation but rather points to their kinship. In clinical practice they are closely interwoven; in a study of a patient, the understanding of one factor is certainly not clear without an understanding of the others. Into each one's practice come a hint of this interrelation and also the desire to master it. We find this concept helpful, not only with the patient whose symptoms have no obvious cause, but particularly with those whose history and findings offer no clue whatever. In taking a history we can at least mention the five subjects to the patient to see if one or more of them may lead to something suggestive. In a discussion of these points the patient often reveals pertinent facts.

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*To be presented at the Midwinter Convention of the Research Study Club, Los Angeles, Calif., January, 1955.

Studies on these five problems are the object of our constant search. The special aim is to find those that suggest an approach to treatment. Rapid strides are being made in each subject, and the hope is that our offerings will provide a glimpse into recent findings, from sources to which our readers ordinarily would not have ready access. We have wandered from the medical library to the bookstore, where authors present medical topics in language understandable to lay readers. Little is to be found in ear, nose and throat literature. It was Socrates himself who advised us — "Employ your time improving yourself by other men's documents. So shall you come easily by what others have labored hard for."

* * *

1. NUTRITION.

Despite the intensive labor that has been expended on the part played by salt in the body economy, we still have much to learn about the real need for it by our tissues. Meneely[1] takes us back to the days when our forebears were cave-dwellers or nomads and lived by hunting and fishing; later came the more settled pattern of life in settlements, with the beginning of agricultural industry and an adjustment of food habits to new conditions. A cereal-vegetable diet in large part replaced the exclusively meat diet. Here came the change in sodium intake; whereas the earlier meat diet contained four or five times as much potassium as sodium, the cereal-vegetable diet had 12 to 20 times as much. This may be an explanation for the use of salt as an added item of diet. By way of corroboration, it is well known that the herbivora travel long distances to reach salt licks and that the carnivora do not. Even the study of word roots in the most ancient tongues provides evidence that the origin of mankind's use of added salt began with the change from nomadic to agricultural existence; the word for salt is found in primitive Greek, but not in Sanskrit. The Sermon on the Mount reveals that salt was held in high regard in Palestine in the days of Jesus: "You are the salt of the earth, but if salt has lost its taste, how shall its saltness be restored?" (Matthew 5:13, Revised Standard Version). The wide-spread longing for salt is not necessarily proof of a real need; Stefansson found in his years in the Arctic among the natives, when he lived their lives and ate their food, that the desire for salt as an added item of food actually disappeared in about three months. Nonetheless modern man continues to add it to his diet, and in certain diseased states may suffer a penalty in edema, heart failure and hypertension. Salt is a dietary essential, but there is an urgent need to re-examine man's requirement for it, its evident relation with potassium intake and its native toxicity. We should continue to unravel and establish as precisely as possible the distinction between taste and need.

To date it appears that the average adult American diet provides from 5 to 10 grams of sodium daily without any from the saltcellar and that this amount in the food itself gives all that is needed. If an excess of salt is taken, it does no harm if it is promptly eliminated. The giving of salt, however, can be very dangerous, in fact in the presence of cardiac or renal dysfunction it may be lethal. Following any surgical procedure producing stress there is a transient physiologic salt retention, because of increased adreno-cortical activity. The administration of salt during this period can result in a dangerous excess of salt in the body.

Many damaging factors are operative in causing deterioration of our foods. In their natural states they promote growth and maintain health, but Knight[3] makes it clear that, by *processing*, they are converted into substances that are of poor quality and may even be harmful to the consumers. Owing to ignorance and the greed which has depleted our forests and soils, farm lands and grazing areas have become impoverished, and so their products are now more or less lacking in important food elements. To protect the growing fruits, vegetables and grain, there is now a wide-spread use of insecticides. These are highly poisonous and carry danger to health in ever increasing degree; moreover they tend to contaminate the soil itself to the point of being destructive to essential organisms living in it. Even though good harvests may still be gathered, the grains are almost always processed — for purposes of preservation, of ease of transport and of readiness for use by the housewife with a minimum of labor — so that they are reduced to a state of shocking impoverishment. By this processing, wheat flour loses its endosperm and coating, and thereby is deprived of much of its minerals, B Complex and vitamin E, as well as some of the natural protein contained in the grain. Another processed food, white sugar, is now consumed in an incredible amount in this country, as much as 125 pounds a year per person; the consumer would be far better off if he adhered to the use of natural carbohydrates and sugars, such as whole grain breads, honey and raw fruits. Recurrent tonsillitis, sinuitis and otitis media respond well to such a regime, plus a high protein intake. To the gross abuse of refined sugar is traceable much of the dental caries, found especially in growing children. Better foods will become available only when the public demands them, and it is the physician, regardless of his specialty, who must guide the public to the knowledge of good nutrition.

Modern milling brought in the white, impoverished flour — with its unquestioned contribution to degenerative disease. For those to whom this fact seems of immediate importance and who preach the value of whole wheat and other whole grains, the study of McCance, Prior and Widdowson[3] will add a feeling of assurance as to the value of B Complex in constipation. The brown bread and the white were fed to normal subjects with a small amount of barium in order to follow the sequence. The brown bread required more chewing and took longer to eat. The subjects preferred the white bread at first but came to like the brown bread more and the white bread less. The brown bread appeared to have a more stimulating effect on the saliva and the gastric juices. The gastric emptying time was less for the brown bread. The residue from the brown bread left the colon 24 hours sooner than that from the white bread, probably owing to the bulk of the brown and to the stimulus of the B Complex, which is rich in the whole wheat but removed from the white. The average passage time through the entire gastrointestinal tract was 2.1 days for the brown bread and 3.1 for the white.

Reduced to simplest terms, the cause of obesity seems to be either too great an intake or faulty metabolism. One frequently sees people even grotesquely obese whose eating habits appear to be not unreasonable. The general opinion is that the moderate amount of food ingested is, owing perhaps to an endocrine lack, not properly burned up and eliminated. In Jolliffe's[4] opinion, however, obesity is invariably caused by a greater intake of calories than is needed. This disproportion is brought about by an abnormal appetite — bulimia. He offers an interesting hypothesis in an effort to explain the phenomenon of appetite. He assumes the presence in the brain of an appetite-regulating mechanism. To popularize the idea, in a book written for the laity he gives it the name "appestat" and represents it as situated in the hypothalamus. This is in line with the present trend of opinion — that the hypothalamus is the controller of many functions, such as the regulation of body temperature, water balance, and sleep. These functions are subject to various disorders, if the centers are damaged by disease; and Jolliffe believes that, like the others, the appetite regulator is vulnerable. He says that 28 per cent of the people in this country are overweight and that, by the age of 50, this is the case in one-third of the men and one-half of the women. Like other habits, that of overeating cannot be changed over night. Old sins cast long shadows.

It seems clear that most overweight persons are not obese as a result of an organic disease and that control of the intake of food is the key problem. Height-weight tables are inadequate and there must be individual evaluation of the patient's physique and history. Why does a given patient overeat and why does he or she wish to reduce? Brosin[5] adopts four categories to explain overeating: *1.* A response to emotional tension; *2.* A substitute gratification in intolerable life situations; *3.* A symptom of emotional illness such as depression and hysteria; and *4.* Addiction to food. It is common for patients to accept a prescribed diet but to cheat without reporting. Social and family pressure favor reducing but often the victim cannot tolerate the loss on account of some inner motivation to be overweight; he might, for example, take this means of avoiding open competition with others, with its accompanying strain and anxiety. Perhaps he wants to go on being lazy. Irritability is fairly often found in patients under a reducing program and if possible good substitutes for the desired food may be furnished by mental and bodily activities.

Obesity is a double handicap to older people; as Keys[6] points out, there is a progressive weakening of their muscles and yet they must move an undue burden of sheer weight. In addition, the so-called degenerative diseases seem very clearly to be promoted by obesity. Nutritional needs of older people, however, are not different in kind but only in amount, from those of younger generations. They need the same foods. The various infirmities associated with old age manifest themselves in different decades, in some persons as early as the 40th year, commonly in the 60's. The basal metabolic rate declines steadily with age, about 3 to 5 per cent a year after age 50. The older person tends to follow the dietary pattern of his community rather than any special pattern of old age itself. If a popular diet in a given region is, for example, conducive to pellagra, then pellagra will frequently be found in the older members of that community. In addition, many are apt to follow the pattern of their earlier years, and not to profit by the advances in nutritional knowledge. Often a fight is necessary to counter the claims of food faddists, purveyors of nostrums and writers who promise miracles from peculiar diets. The primary means of improving nutrition, even with the aged, must be education.

Today, patients are undergoing operations under circumstances in which they would have been declared inoperable only a few years ago. This puts an extra burden on the teams which supply nourishment to the patient and Rhoades[7] states that a good regimen often tips the scale toward recovery in patients who would otherwise be lost. After severe burns the amount of "C" which must be administered to produce an increase in the output of "C" is very great indeed — of the order of 1000 to 2000 mg. per day; furthermore, we have learned that the "C" content of the adrenal is reduced. It is best to give very large amounts of "C" for several days and then to continue with fairly large amounts. The "B" vitamins are important in regulating gastrointestinal function and should be supplied for a period after gastrectomy. "B_1" deficiency is apt to develop during fever and hyperthyroidism; during a thyroid storm, the requirement may go up at least fourfold. "B_2" is apparently deposited in the body during periods of protein synthesis and excreted during periods of protein breakdown.

During World War II large numbers of Danes, who had taken part in the resistance movement, were sent to concentration camps in Germany. After the war 1300 were repatriated. These men have been carefully examined from time to time, to determine their right to compensation, and Hermann and Thygesen[8] review the latest findings in 120 of them. Lost weight was seldom regained, amounting to one-fifth of the original weight in a few cases. In some, hunger dystrophy from malnutrition caused irreversible changes. Mental deterioration showed itself in impairment of memory and inability to concentrate; the memory of 70 of the men was so enfeebled that they had to write down whatever they wished to remember. In more than one-quarter of those who suffered mental effects, working capacity was found to be progressively impaired.

Although our chief aim is to present what tends to improve nutrition, it is not unfitting to see what can happen from the misuse of one of the great factors, vitamin A. A patient, whose case is described by Gerber, Raab and Sobel,[9] when in her 20's began the use of "A" to combat ichthyosis and continued to take it for nine years in prodigious amounts, reaching 500,000 units daily. Her suffering from effects of this record-making ingestion of "A" led her to five hospitals; she had severe headache, serious eye trouble in the form of diplopia and blurred vision, skeletal rigidity, decalcification of some bones and exostoses of others, and incontinence of urine. Her headache was of a degree to lead to a diagnosis of serous meningitis and a right subtemporal decompression was performed. Her course was ever downward. Only on her last admission was the significance of her "A" addiction recognized; her fasting blood level of "A" was about 40 times the normal. Withdrawal of "A," except as part of the usual diet, was followed in a few weeks by marked improvement — entire loss of pain, clearing of her intractable itching, restoration of vision, loss of diplopia and the return of more nearly normal posture and freedom of movement.

An obvious inability of the geriatric (or any) patient with achlorhydria to digest, absorb and retain proteins prompted Sharp[10] to determine a person's ability to absorb protein through the use of a tagged protein feeding material — a preparation of yeast which had been tagged with nitrogen. In a carefully controlled study, patients were hospitalized and their excreta examined spectrographically. Lack of protein absorption in the geriatric patient is one of the most important causes of senile tissue changes, particularly of the skin and mucous membrane. Abnormal states in these tissues may well have a bearing on tumor formation. This study has also suggested a possible method in stopping the growth of human cancer — by "induced abnormal protein synthesis." Complex chains of organic matter make up protoplasm, which is the name given to "living" tissue. Protein elements are the main constituents of these chains. When growth or repair is required, these complexes must be duplicated. This process is called protein synthesis. The current concept of the mechanism of this process is that when a cell manufactures a new protein complex, either for growth or for repair, an already-existing chain of protein elements must serve as a model, or "template," for the orderly construction of the new, or duplicate chain. Thus nature provides a "jig" for the mass production of the complicated sub-assemblies that make up life. The plan behind the experiment is that a tumor can be induced to assimilate a protein sub-element which has been distorted by the introduction into its chemical structure of an *unnatural* amino acid. The template, or jig, thus produced within the tumor cell would not then readily serve as a model for the construction of further protein elements made of the natural nutrients found in the human body. Thus the structure of material within the tumor cell could be so altered that it would lose its capacity to reproduce — and in this way its growth might be stopped.

In his account of experiences as Senior Medical Officer of a large whaling fleet in the South Atlantic, Robertson[11] makes a contribution to our knowledge of nutritional sources, purely incidental to his general tale. Among the products of the whaling industry are soap, magarine, cattlecake, fertilizer that enriches our wheat, feed for chickens, vitamins, glaze on photographs, oil for watches, the tan on our belts, as well as 70 different pharmaceutical preparations in common use

The study of nutrition has at last caught the attentio many in the medical profession and there is no questio to the interest shown by the patient, who has a deep conc in getting advice about what he should eat and what he sho avoid. This interest on the part of the public finds a respon in popular magazines. In an article written for the lay reade "Bread from the Sea," Davidson[12] narrates the story of a intensive study in many parts of the world of the unicellula alga as a potential, already an actual, source of food. From the University of Maryland, an estimate of land productivity for three important foods furnishes these data: In one year, per acre, beef production is less than 250 pounds, soy beans less than half a ton and algae 40 tons. Thailand collects plankton — algae and other small marine life — from adjacent seas, a harvest of 5000 tons a year. Japan's leader in this study, Hiroshi Tamiya, at the Carnegie Laboratory at Stanford University, recently demonstrated the suitability of algae for food in many acceptable forms. They taste good, too. The University of Texas has done important work in the selection of particular species of algae. Their rate of growth is amazingly rapid and the product is of high value in proteins, fats, starches, vitamins — in fact every food component needed to sustain life. Sunlight, carbon dioxide and minerals found in soil are worked upon in this microscopic food factory at small cost. Like all protein foods, the output spoils easily and for this reason it is usually dried to a fine powder which can be stored indefinitely. In Venezuela in a leper hospital patients were fed for four years with "plankton soup" in the hope of its having some curative value; while this hope was not borne out it proved to be a good food. In this study there is a reasonable prospect that the hungry areas of the world will find the solution for their problems of food shortage at small expense.

To keep pace with the increase in world population, this pressing need of increasing food production was the subject of discussion in the House of Lords[13] recently. Only 100 years ago the world population was a mere billion. Now it is two and one-half billion and in 70 years will probably double itself to five billion. Areas under cultivation must be extended and new fields of production explored. Plankton can be harvested in unlimited amounts from the sea and become an important source of protein and vitamins. Chlorella, a green alga, is already under scrutiny in the United States as a rich source of vegetable protein. Of course, when any radical change in a nation's habits is proposed, social, religious and economic obstacles are to be looked for, especially in some Eastern countries.

2. ENDOCRINE.

Stating that the recent advances in Endocrinology have markedly changed our concepts, Goldzieher and Goldzieher[14] have brought out a book to which many scholars have contributed. All speak of the wealth of new information and the variety of potent therapeutic agents. Until recently endocrine therapy has been considered useful when it overcame a deficiency; unless a deficiency was demonstrable, the use of hormones was considered to be unwarranted. This has been the prevailing viewpoint, even though everyone, especially the otolaryngologist, has been familiar with the local use of epinephrine; also all have known of the use of insulin in the treatment of certain mental diseases; however, with the advent of cortisone and ACTH there has been a radical change in attitude. These hormones are now used freely in a great variety of diseases in which there is no demonstrable hormone deficiency at all. In fact, cortisone and ACTH are in common use on an empirical basis, like quinine or the salicylates. Hormones are known to alter or influence specific phases of metabolism; if such changes are desirable, a hormone is indicated. For example, if one intends to promote an increase in anabolism (protein synthesis, formation of new tissue), androgenic steroids may be used, without regard to the patient's sex or gonadal status. The contributors to this book, in their respective fields, draw on special experience; in the following paragraphs, examples give an idea of its extent and worth.

Shelton and Skeels[15] note that a spurt in growth of three to four inches is usual at puberty, when primary and secondary sex characteristics begin to develop because of the increase in the secretion of gonadal and adrenal steroid hormones. Skeletal growth usually terminates by the 17th year in the female, while in the male it may continue to the 21st year. Underfed children fail to grow normally, but they are only moderately shorter than the average. A high caloric, high protein diet is at once essential. Testosterone in small doses is probably the best anabolic stimulant. In boys a dose of 10-15 mg. daily is often sufficient to produce remarkable nitrogen storage and linear growth. If the judicious use of testosterone for two or three months is alternated with similar periods of rest from treatment, there is little danger of causing premature epiphyseal fusion. Serial "bone age" determinations should be performed, and the androgens should be discontinued when the bone age is brought up to normal. In girls, androgens, if used at all, should be employed cautiously; the danger of irreversible virilization is always present; however, estrogenic preparations given concomitantly usually obviate or at least minimize the untoward androgenic effect. Small doses of globin insulin frequently stimulate the appetite. * * * As to the thyroid, most hypothyroid children are not frankly obese, and it is perhaps for this reason that many cases are overlooked. Bone age is retarded, especially in the hips. Mild cases may be difficult to diagnose; however, if the serum cholesterol is elevated and then falls 30 per cent and stays down during thyroid administration, the diagnosis is established. Rapid development can occur after adequate treatment of the severely hypothyroid child.

Senescence on the other hand presents a very different picture. The problem of aging seems to Goldzieher and Goldzieher[14] to have two aspects: The inevitable changes and also those that are amenable to medical approach, as in any other degenerative disease. The advanced state of senescence may be out of all proportion to the age of the patient; the removal of debilities may lengthen life. The few facts available indicate that aging is associated with chemical changes which affect the whole body and all of its cells. Studies with isotopes have shown that throughout life the cells turn over their atomic constituents far more rapidly and more completely than one had imagined. In addition to the turnover on the atomic level, a continuous and rapid change also takes place on the molecular level, in the two antagonistic phases of catabolism and anabolism. In the young these two processes are well balanced; in old age, there is a definite decline of both, but the decline of anabolism predominates. The chemical constituents are not replaced at the same rate as before. The difference is not only in speed but in quantity — hence the body substance decreases. It is well-known that the size of the heart, liver and kidneys decreases in the aged. The thyroid and the gonads decrease in size and in secretion; the function of the pituitary and adrenal glands is diminished to a lesser degree. The decline of testicular function is confirmed by the decreased urinary excretion of the 17-ketosteroids. About one-third of the 17-ketosteroids come from the testicle and two-thirds from the adrenal cortex. Testosterone and, to a less extent, estrogen retain tissue nitrogen and help to build proteins. Both retain water in the tissues and affect the calcium metabolism and the mineral content of the skeletal system. Even the central nervous system is under the influence of the gonadal hormones and strongly reacts to their absence. It would seem that the prescribing of androgen ought to be of value in arresting the process of decline; the soundness of this assumption seems to be corrobated by clinical experience. Giving of androgen, especially testosterone propionate, in the aged, both male and female, causes a prompt increase in body weight, a gain in strength and vigor and a sensation of well-being. In the female, testosterone must always be accompanied by a commensurate amount of estrogen to prevent symptoms of masculinization; the dosage of the testosterone must be lower than in the male — usually one-half the amount. On the other hand, the amount of estrogen should be increased over that given to the male. Special geriatric preparations now provide estrogen-androgen doses combined with vitamin and mineral supplements. Hormone therapy in senescence cannot exert its best effect unless the nutritional requirements are adequately maintained. It is often better to advocate frequent small meals than fewer large ones. An adequate intake of calcium is desirable in view of the tendency to develop osteoporosis in the aged. Milk is an excellent source but if the necessary one or two glasses of milk a day are not consumed a substitution can be made by using one of the common calcium preparations. The response to adreno-cortical extract is better if given jointly with substantial amounts of vitamin C and the B complex.

3. AUTONOMIC.

Since the human body is a unified organism, it cannot have several independent master controls. The endocrine axis has its many actions controlled from the pituitary. In turn the target glands modify the activity of the pituitary. But where is the primary instigator of pituitary function? One school of endocrinologists has suggested that the system is self-sufficient, without domination or control by any mediator outside its own circle; however, the principle of unity is once more confirmed by recent work that integrates the humoral (endocrine) and neural adaptive mechanisms of the body.

The basal nuclei of the brain principally found in the hypothalamic area, have for a long time been recognized as the control center for neuromuscular, neurovascular and neurosecretory functions. Porter[16] has now demonstrated changes in electrical activity in the hypothalamus in response to systemic stress.

When ACTH stimulates the adrenal cortex, circulating eosinophiles are markedly reduced in number; this is the basis of the well-known Thorn test. Hume[17] has shown that electrical stimulation of the hypothalamic areas induces eosinopenia, suggesting activation of the pituitary to produce ACTH. Production of eosinopenia by pituitary response to adrenalin injection can be prevented, if these hypothalamic areas are detroyed. These experiments demonstrate, then, that an intact hypothalamus — particularly the posterior portion — is essential to the production of normal endocrine (pituitary — adrenal) response to stress. Thus the endocrine system is not independently activated; its function can be stimulated or prevented through basal nuclear (hypothalamic) action. Hume also suggests that afferent somatic and autonomic nerves coming from the region of body stress may activate the basal nuclei to drive the pituitary into action. He also suggests that chemical substances produced at the point of body injury (stress) may be carried through the circulation to the hypoon the molecular level, in the two antagonistic phases of catabolism and anabolism. In the young these two processes are well balanced; in old age, there is a definite decline of both, but the decline of anabolism predominates. The chemical constituents are not replaced at the same rate as before. The difference is not only in speed but in quantity — hence the body substance decreases. It is well-known that the size of the heart, liver and kidneys decreases in the aged. The thyroid and the gonads decrease in size and in secretion; the *function of the pituitary and adrenal glands is diminished to* a lesser degree. The decline of testicular function is confirmed by the decreased urinary excretion of the 17-ketosteroids. About one-third of the 17-ketosteroids come from the testicle and two-thirds from the adrenal cortex. Testosterone and, to a less extent, estrogen retain tissue nitrogen and help to build proteins. Both retain water in the tissues and affect the calcium metabolism and the mineral content of the skeletal system. Even the central nervous system is under the influence of the gonadal hormones and strongly reacts to their absence. It would seem that the prescribing of androgen *ought to be of value in arresting the process of decline;* the soundness of this assumption seems to be corrobated by clinical experience. Giving of androgen, especially testosterone propionate, in the aged, both male and female, causes a prompt increase in body weight, a gain in strength and vigor and a sensation of well-being. In the female, testosterone must always be accompanied by a commensurate amount of estrogen to prevent symptoms of masculinization; the dosage of the testosterone must be lower than in the male — usually one-half the amount. On the other hand, the amount of estrogen should be increased over that given to the male. Special geriatric preparations now provide estrogen-androgen doses combined with vitamin and mineral supplements. Hormone therapy in senescence cannot exert its best effect unless the nutritional requirements are adequately maintained. It is often better to advocate frequent small meals than fewer large ones. An adequate intake of calcium is desirable in view of the tendency to develop osteoporosis in the aged. Milk is an excellent source but if the necessary one or two glasses of milk a day are not consumed a substitution can be made by using one of the common calcium preparations. The response to adreno-cortical extract is better if given jointly with substantial amounts of vitamin C and the B complex.

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Porter[18,19] has further shown the intimacy of the autonomic centers and the pituitary axis, by demonstrating that adrenal hormones can modify the responsiveness of the hypothalamic areas to stressful stimuli.

Thus, the higher emotional centers, the autonomic centers of the hypothalamus and the pituitary-governed endocrine system *cooperate* to effect humoral and neural control. The basal nuclear area is the central switchboard. Autonomic dysfunction and endocrine imbalance are integrated disturbances, so their clinical similarity is understandable.

4. STRESS.

In opening a symposium on Stress, shared by other recognized leaders in this field, Selye[20] reviews his work of the past 18 years. The body's response to stress, caused by a wide variety of factors, is specific when it combats infection, trauma and the like; it is non-specific when (to borrow the language of the physicist) the body is subjected to pressure and tension. Selye confines his discussion to the latter conditions. During the reaction to pressure or tension, the outstanding manifestations are adreno-cortical enlargement in contrast to involution or degeneration of other organs; in fact, the adrenal cortex appears actually to flourish on stress. This useful role in the body's non-specific adaptation is what Selye calls the "alarm reaction." The general adaptation syndrome comprises three distinct stages: *1.* The alarm reaction; *2.* The stage of resistance; and *3.* The stage of exhaustion. His concept is that there are many diseases that have no single cause, no specific pathogen, but are largely due to non-specific stress. His early work was carried out with crude glandular extracts; later research by many workers, notably the classical investigations of Kendall and Reichstein, identified and purified the many hormones involved and defined their functions. Selye's introduction is followed by studies of stress reactions observed by the other contributors; these relate to the various body systems and to the problems that confront the pediatrist, the psychiatrist and the clergyman. One contributor lightens an otherwise somewhat ponderous subject, to extol the Importance of Leisure; Ogilvie[30] unashamedly narrates how, in his schooldays in England, his father expressed concern lest he be overworking, but his anxiety was allayed by the headmaster's assurance that "your son has a remarkably well-developed protective instinct.' The lesson learned from long experience is that "the best way to prevent stress is not to suffer it." Man, alas, knows too much for his peace of mind. When danger is upon him, he faces it and calls up all the autonomic and endocrine mechanisms that can protect him; but, unhappily, he is not always able to dismiss them when the need is over.

In a well-planned endeavor, seven physicians in general practice, Finlay, Gillison, Hart, Mason, Mond, Page and O'Neill[31] assemble their experiences in "stress disorders"; their fields of practice range from the congested areas of London to the rural communities of England. About one-fifth of all patients seen on any one day in urban practice suffer from stress and even in the country the proportion is one to ten. The greatest difficulty in appraising the many factors that combine to cause symptoms is, in their panel practice, the lack of sufficient time; nonetheless this symposium represents an agreement on diagnosis and classification, reached only after careful discussions. In the experience of Gillison, there are three classes of stress disorders: 1. Phobias, such as fear of tuberculosis or cancer; 2. High strung personalities, with inner conflicts but no particular anxiety revealed; and 3. What may be called "moral stress." Mason finds stress syndromes to be common, such as tiredness, headache, fear of cancer or heart trouble or appendicitis, seborrhoea, peptic ulcer, localized pain in this or that region, diarrhoea and night sweats. This study indicates that of all the different causes of unhappiness and inefficiency, psychosomatic illnesses are probably the greatest.

In their book, Holmes, Goodell, Wolf and Wolff[32] give an thalamus and thus stimulate the basal nuclei to prod the pituitary. He contends also that neural pathways (association bundles) from higher cortical centers can activate the hypothalamus to the production of pituitary axis activity. For example, electrical stimulation of the prefrontal cortex can produce eosinopenia.

Porter[18,19] has further shown the intimacy of the autonomic centers and the pituitary axis, by demonstrating that adrenal hormones can modify the responsiveness of the hypothalamic areas to stressful stimuli.

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In their book, Holmes, Goodell, Wolf and Wolff[22] give an exhaustive study of the functions of the nose, particularly of the erectile tissue of the turbinates and of the tubular serous glands which abound in the lining membrane. Color changes of the mucosa are evidence of variations in the flow of blood and vary from pale yellow-red to deep cardinal. They may accompany bodily exposure to temperature changes such as chilling or overheating. They loom large in the special field of external psychic pressures; local reactions in the lining membrane of the nose result from a wide range of such emotional tensions. The authors present very detailed histories of their patients and their individual nasal reactions to anxiety, remorse, frustrations, social and familial maladjustments and conflicts, tension in business relations. Nasal obstruction, in fact, constitutes a part of a defense reaction against unwelcome or threatening situations. This demonstrates that the pathogenesis and the treatment of chronic nasal congestion with which we deal every day, present a medical problem in a very broad sense. Rather than limit our effort to what Pierce[23] called "manicuring the nose," we recognize that these conditions respond in the long run to treatment based upon a discerning diagnosis.

5. ALLERGY.

Tantaque in his rebus distantia differitasque
*Ut quod ali cibus est aliis fuat acre venenum.**

The old aphorism, "One man's meat is another man's poison" can be traced to Lucretius[24] in the first century B.C. True appreciation of the importance of inhalants took shape in the 16th and 17th centuries. Brown[25] pays a high tribute to the shrewd observations of Morell MacKenzie, who in 1854 laid the blame for seasonal hay fever on pollen, for asthma on lycopodium and animal emanations, for coryza on house dust, tobacco smoke and grease fumes. MacKenzie's "idiosyncrasy" became in 1906 von Pirquet's "allergy," although the underlying problem remained unsolved. Even as late as 1905 most physicians believed allergic disorders to be due to bacteria. An early champion in the fight against pollen sensitivity was Brown's own teacher, John Freeman of London, who in 1911 [illegible] se of injections of timothy pollen extract for hay [illegible] Manifestations of allergy in fields other than resp[illegible] came better and better recognized in the latter ha[illegible] century. Angioneurotic edema was defined by [illegible] Quincke and others; anaphylaxis by Flexner and [illegible] allergies by many observers, especially during [illegible] years; atopic eczema by Walker in 1917. Ep[illegible] first used in 1903 as a means of controlling br[illegible] ephedrine came into general use in 1917 [illegible] the many synthetic compounds; in 1[illegible] in was introduced; the antihistaminics in 1[illegible] recently, ACTH and cortisone. Brown specu[illegible] major discoveries are still to be made and wo[illegible] happen to Allergy in the next 20 years.

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* In these matters there are differences so great that what is food for one man is for others rank poison.

It is important to the physician to detect if possible which of his patients have allergic sensitivity, either as a major factor or as a minor and it is estimated by Kern[24] that of all patients, one in seven will at some time have a major allergic disease, and one in four will have a less severe, probably more obscure allergic reaction. Evidently, allergy covers a large part of any medical practice. Allergy may give rise to a host of symptoms and in many parts of the body. In the same patient, sensitivity to one allergen may diminish and reaction to another may develop. Allergy may affect one organ at one time and another at another time. These patients do not inherit an allergic disease but a capacity to be sensitized to things more easily than are other persons. They are born that way and remain so until they die. The defect does not change; it is the sensitivity pattern that changes. Old sensitivities may be lost and new ones may develop. Different parts of the body may become new shock organs, while parts once sensitive may lose their reactivity. The number and potency of allergens are on the increase, as found in the use of many of our new and most helpful remedies, such as certain antibiotics. The best way to learn which patients are sensitive is simply to ask them. "It takes the women to remember that a great-aunt got hives from Jerusalem artichoke." If the family history is positive, the patient could be aller[illegible] the personal history is positive, he is allergic. Eosinophilia suggests allergy; its absence does not rule it out. The syndrome of Meniere includes a fair proportion of allergic nature; the middle ear also may suffer from allergic edema. Psychic factors may pull the trigger, but they do not load the gun; one does not see an asthmatic in whom only psychic factors are present. Except in rare instances, the allergic factor is preponderant. For asthma, one should not give drugs that cut down an already scant sputum, such as atropin and antihistaminics; doing so can cause death. Opiates, unwisely given, are still the commonest cause of death in asthma. All chemotherapeutic agents are potential allergens. Each one as it appeared was hailed as low in toxicity and of neglible allergenic significance, only to prove quite the reverse when lapse of time had permitted its second and later courses in patients. Penicillin is no exception; a score or more of sudden deaths have been reported. Often, if the patient demands an antibiotic for a minor ailment, one may mollify him by saying, "Don't shoot sparrows with a 16-inch gun." If the reaction to a drug persists unduly after its use is stopped, and especially if there develops a rising eosinophilia, there is the threat of periarteritis nodosa; this calls for the immediate use of cortisone. Corticotropin and cortisone are only palliative in allergic states; there is no diminution in the underlying sensitivity. Prolonged use of corticotropin is followed by hypoplasia of the pituitary and hypertrophy of the adrenal glands. When treatment is stopped, the pituitary regains some, but not all of its loss after months, while the adrenal glands shrink to less than their former size. Prolonged administration of either drug leaves the patient worse off than when he started.

Twenty-three allergens are named by Sheldon, Mathews and Lovell[27] as their choice for routine survey. They give a solemn warning of the dangers of the intracutaneous procedure and dwell on the absolute need of a prior scratch or prick testing; eight allergens are properly called "explosive," because they have caused fatalities when used intracutaneously — mustard, cottonseed, flaxseed, ginger, buckwheat, mushroom, sea food and nut meats. All the deaths occurred when the intracutaneous tests were performed without prior scratch or prick testing. Sensitivity to a food can often be determined merely by attention to a patient's story; in cases where the history is not clarifying, one can best depend upon the trial-and-error method — elimination diets. In general, skin tests are not reliable for foods.

Reactions from penicillin have been reported from several sources in 1954. Farber, Ross and Stephens[18] sent out questionnaires to California physicians and received over 1000 replies; the data thus gathered show more than 520 moderate reactions to penicillin and over 300 severe reactions with 7 of them fatal. The antibiotic is believed to unite with the serum proteins to form antigens; the patient is thus rendered vulnerable to a subsequent injection of the antibiotic which sets free in the blood stream histamine or histamine-like substances, and the end result is shock. Experimentally, ACTH has been used to avert shock in guinea pigs; given one hour before injection of an antigen, it diminishes or inhibits shock, but, if given when a reaction is manifest, it is valueless. For the past two years Farber and his associates have combined with the antibiotics one of the antihistaminic drugs — benadryl, pyribenzamine, histadyl or chlortrimeton. In more than 3000 injections no severe anaphylactic reaction has been observed. Evidence of shock calls for vigorous handling without delay — epinephrine by intramuscular injection, an antihistaminic drug by intravenous route and oxygen administered under pressure to avert pulmonary edema. One of us (J.A.H.) finds that prolonged infusion of hydrocortone in an intravenous vehicle is also effective and may prove superior to any of the others.

That alarming reactions can follow the use of even minute amounts of penicillin is shown by Carter and Cope.[19] In 1946 and 1953, a nurse had had injections of the drug for congenital syphilis and bronchitis. After the second event she noticed wheezing and later a rash. Three months after this second treatment she merely applied penicillin ophthalmic ointment to one eye. A reaction followed speedily — shortness of breath, flushing of the face and abdominal cramps. However, epinephrine, phenobarbital, atropin and pyribenzamine brought relief of the symptoms.

The Chief Medical Examiner of New York City lists eight cases in which the injection of penicillin was followed by sudden death. In no instance was there evidence that the drug had entered a blood vessel. Rosenthal[30] pleads for discrimination in its use and a careful search for a history of any previous unfavorable reaction, pointing out that reactions occur after the use of penicillin by any route, not only the intramuscular.

A helpful classification of allergic diseases is offered by Coca,[31] who considers that five categories are distinguishable. *1. Atopy.* This group includes bronchial asthma, hay fever and infantile eczema; it is the hereditary character which unites these conditions in one group and separates them from the other categories. *2. Allergy of infection.* There is a lack of the familial character; the sensitivity is established merely by the infection, such as the pathogenic fungi and the viruses. *3. Serum disease.* This is regarded by many as the clearest example of anaphylaxis, a specific reaction in which injected foreign serum becomes an antigen. *4. Contact dermatitis.* The familiar example is that caused by poison ivy. The numerous excitants of this condition are all non-antigens; no protein excitant has been encountered. The sensitivity seems to be limited to the cells of the epidermis; the intracutaneous test with the excitant results negatively. The oily excitant of poison ivy has been extracted and solutions of it given, by injection and orally, in an attempt to bring about a lessened sensitivity. *5. Idioblapsis; familial, non-reaginic food-allergy.* This is the unusual approach offered by Coca. To signify a food-allergy in which antibodies (reagins) can not be demonstrated and in which food extracts do not produce skin reactions, he adopts the word "idioblapsis" (Greek, *idio,* personal and *blapsis,* a spoiling or damaging). Tachycardia, following ingestion of a food, is the specific reaction of this "familial, non-reaginic" allergy; Coca affirms that this is a means of positive identification of a food allergen. The procedure he has adopted calls for a pulse-record for a few days before the test; the pulse rate is recorded before rising, before each meal, and three times after each meal at 30-minute intervals. The foods to which most allergy patients are sensitive are wheat, milk, egg, potato and meat. Each test calls for only one article of food at a time — small meals, five in one day.

If relative tachycardia occurs, it is of course necessary to wait for its cessation before proceeding with another test. Being one of the most valuable foods, milk is one of the first to be tested; if no sensitivity is shown, milk may be taken freely with a food subsequently tried. In this way, one food after another may be tested, one at a time.

In a lay magazine Cronin[32] gives a useful summary of the problems of allergy. Except for his definition, "hypersensitivity of the body cells to various substances, otherwise normal, which act as irritants on particular individuals," he uses medical terms sparingly yet covers the field well. He gives due place to reactions associated with psychosomatic factors. He cites cases of allergic reactions to foods, inhalants, clothing, even glue on postage stamps. A striking instance is presented of a priest who for six years had had periodic attacks of sneezing and coughing which at times would develop into a two-minute fit of choking; wine which the priest drank at Mass proved to be the excitant, and attempts to give him tolerance for wine failed.

GENERAL.

In our specialty there are many who feel that we should grasp any opportunity to catch a glimpse at what is going on in modern medicine. As Garland[33] expresses it, medical progress has surged onward through the ages, despite such delightful lapses into complacency as that of Ambroise Paré — he who dressed the wound that God might heal it — when he wrote in 1575: "God is my witness * * * that I have labored * * * to throw light on the art of Surgery and bring it to perfection. And in this labour I have striven so hard * * * that the ancients have nought wherein to excel us, save the discovery of first principles; and posterity will not be able to surpass us * * * save by some additions, such as are easily made to things already discovered."

In a survey such as this, there is an immediate satisfaction when we can provide facts and "prescriptions" that may be promptly applied in the office and the hospital. However, such articles are not found in abundance; besides, surely there is information, not yet applicable to practice, with which we would do well to become familiar. Many of the following articles have no immediate bearing on clinical practice, but they are made available for the earnest student interested in basic facts that affect the health of the structures with which we are directly concerned.

Progress may come from suggestions that at the moment seem to lead nowhere. Such an idea is in line with what DuBridge[34] so neatly calls the importance of being *impractical*. Although he holds a torch for knowledge for its own sake, he particularly exults that sooner or later it becomes applied in a most practical way. With the use of steam came the Age of Power; lumps of coal then became man's "black slaves." For ages the world did not change much fróm one generation to the next; now it hardly stays recognizable for ten years at a stretch. To DuBridge, men had not become more intelligent, but had found a new process of thinking, by observing, deducing and predicting. It is called "Science." A searcher in the chemistry of living things might not be working on a cure of any disease at all, but yet, to his surprise, make a great discovery — penicillin — helpful in many diseases. As Louis Pasteur expressed it, "In the fields of observation, chance favours only the mind which is prepared."

Some day we shall better understand the nature of the protein, its function as a bulwark against infection, the uses of the isotopes, how a cell can reproduce itself, and the complete story of a virus.

* * *

Knowledge of life processes has increased during this century, but many signs indicate that a golden age is only now beginning.[35] Chemical biology has already produced or identified the physiologically useful vitamins, hormones and antibiotics. It has brought the theory of the gene to the point where we understand the basic differences between living and non-living systems. In 1951, Pauling and Corey[36] reported the atomic structure of proteins found in bone, muscle and red blood cells. Proteins are responsible for many of the activities that we associate primarily with living things; in this respect they may truly be considered more important than any of the other components of living cells. The major components of skin and muscle are proteins; so are the major components of hemoglobin and the antibodies found in the blood. The first straight advance toward an understanding of protein structure was made in 1900 when Emil Fischer found that a protein molecule is composed of simpler substances, amino acids.

How *proteins are put together from the elements is still* a baffling problem and no one can bring to us the full meaning of the proteins better than Albrecht.[37,38,39,40] It seems to him that we have given little thought to the concept that plants are struggling to *make* their necessary proteins, just as animals are ranging far and wide to *collect* theirs, and that *healthy man eats food of all types — in facts needs to be* highly omnivorous, and that all these efforts have the purpose of making sure that each form of life is getting the required amino acids. Much has been learned about the chemical structure of proteins and yet we do not know how nature starts with the separate elements — carbon, hydrogen, oxygen and *nitrogen — and builds an amino acid.* Of the *amino acids,* eight are essential for man if he is to live. How sulphur and phosphorus are put into protein molecules is another unsolved mystery. We are able to separate a protein into its amino acids and then further separate them into their chemical elements, and yet we have found no clue to the processes by which nature puts them together to *produce* a protein. Plants first make carbohydrates — by photosynthesis; then, if the necessary elements are available in the soil, they apparently convert the carbohydrates into amino acids — by biosynthesis. The amino acids then hook together to make proteins.

The body's store of protein constitutes a bulwark against *infection.* Depletion of this store of protein increases the danger. Cannon[41] finds that rats and rabbits on diets that severely reduce the protein, exhibit a decreased capacity to form specific antibodies. These animals, however, quickly regain their normal capacity to synthesize antibody-proteins when fed a *diet containing* an adequate amount of high-quality protein. Protein-deficient animals display a decreased resistance to experimental infection; there is a marked atrophy of bone marrow. For such depletion of the protein stores and for such diminished functioning of the tissues producing antibodies, a considerable period of time is required. In the broad field of communicable disease it is *acquired* immunity which decides the outcome of any grave infection. Thus the immunity of the newborn is due not so much to the forces of natural resistance as to the transmission of protective antibodies from the mother. From a few months after birth and throughout life, defense against infection depends largely upon one's ability to acquire immunity. Immunity to infectious disease is acquired by recovery from the disease, or by vaccination, or by subclinical doses of the causative agent. Acquired immunity, however, is always relative and tends to vary in degree in accordance with the capacity of the tissues. Antibodies cannot arise, presumably, if there is a lack of available building material, or if there is deterioration of the cells which produce them. Antibody-production may be influenced by the availability either of constructional materials or of activating agents, such as the enzymes, vitamins and salts. Furthermore in the course of infection, specific antibodies continuously combine with a multiplying antigen or its products, become expended, and need to be renewed as the infection progresses. Consequently, in order to function adequately, immune mechanisms must be able to fabricate antibody-protein uninterruptedly. In other words, the overall process of antibody formation must be looked upon as dynamic and progressive. The primary absence of a vitamin may block the process of antibody formation even though the amino acids necessary for the synthesis are available.

Studying the relationship, Cannon[42] also speaks of a nicely balanced or steady state, a dynamic equilibrium, between the protein in the tissues and the protein in the plasma. It is well known that during digestion the amino acid concentration of the blood may show a considerable rise, followed by the rapid removal and destruction of these amino acids by the liver. Some amino acids, however, pass through the liver; and in the fasting animal the amino acid concentration in the tissues may be as much as ten times that in the blood plasma. Soon, however, the concentration reverts to its regular level, as equilibrium with the blood is re-established.

Dulbecco and Vogt[35,43] have developed a technic for studying viruses that attack animal tissue — those that cause smallpox, herpes, influenza and poliomyelitis. Formerly, they could be studied only by the slow and expensive process of infecting monkeys or chicken embryos. The new technic is to grow a single layer of animal tissue cells in a nutrient medium in a small dish and then to add a liquid containing virus particles in a weak suspension. As each particle of virus attacks the tissue cell it will multiply and produce a small visible area of dead cells. Thus the number of virus particles in the original suspension can be counted and the progeny from any single virus can be "bred" for further study. By applying this technic it became possible for the first time to isolate genetically pure strains of each of the three known types of polio virus. This means that it is now possible to start intensive studies of the development and hereditary properties of each virus.

How does a living cell reproduce itself? Commoner[14] conducted an experimental attack on this key problem in biology. What is this biochemical process — the fundamental basis of all reproduction? Tobacco mosaic virus offers special advantages for his experiment. When a very minute amount is inoculated into a tobacco leaf, large amounts of new virus soon appear. The flowers produced are streaked, instead of showing normal solid color; the shape and the color of the leaves are also affected. This particular virus can be taken out of the infected cell and isolated as a pure single substance, a nucleoprotein. It can be kept in solution for years and, most important, will still be capable of reproducing itself if put back into a tobacco cell. In brief, it can be taken out of the cell, handled as a mere chemical, and later put back into a new cell without losing its biologic powers. The fundamental approach was to compare the biochemical process in otherwise identical pieces of infected and uninfected tobacco leaf. It was found that the new virus formed in an infected cell is made from the simplest form of nitrogen, ammonia.

Being Director of the Isotopes Division of the Atomic Energy Commission at Oak Ridge, Tennessee, Abersold[15] can speak of isotopes with authority. He reviews the history of their medical use, from the discovery by Curie and Joliot that ordinary elements can be rendered radioactive, to the present time when 300 labeled compounds are available. He lists six now in common use for diagnosis and treatment; radioiodine 131 and radiophosphorus 32 are the ones most widely utilized. Recent developments in technics and instrumentation have materially advanced the exactness of localization; he cites the use of diiodofluorescein in the mapping of the boundary of brain tumors. In therapeutic applications, on the contrary, radioisotopes are used only as an adjunct to other means of radiotherapy or chemotherapy. As tracer atoms, however, radioisotopes are unique. It seems certain that their extended use will contribute richly to medical advancement.

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MEETING OF THE GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The Georgia Society of Ophthalmology and Otolaryngology will meet March 11 - 12, 1955, at the General Oglethorpe Hotel, Savannah, Georgia.

The speakers will be: Dr. Francis H. Adler, Philadelphia; J. W. McCall, Cleveland; Dr. J. A. Hilger, St. Paul; Dr. Walter H. Fink, Minneapolis; Dr. James H. Allen, New Orleans; and Dr. P. E. Ireland, Toronto, Canada. For further details, address Alton V. Hallum, M.D., Secretary [illegible] Doctors Bldg., Atlanta 3, Ga.

AN EVALUATION OF CERTAIN THERAPEUTIC AGENTS AND PROCEDURES IN THE TREATMENT OF ACUTE DIFFUSE EXTERNAL OTITIS.*

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INTRODUCTION.

Otitis externa is an acknowledged problem of military personnel serving in tropical or semi-tropical climates. It is particularly disabling to those whose duties require the use of ear phones. Thus, acute diffuse otitis externa, the most prevalent variety, has been of special concern to the Air Force, and the study of this disease has had active support from that Service.

Early investigators believed that diffuse external otitis,[1] variously termed hot weather ear, desquamative external otitis, fungous ear, tropical otitis externa, and furunculosis, was due to a mycotic infection. Later, Greaves,[2] Syverton, *et. al.*,[3] Senturia,[4] Singer, *et. al.*,[5] and others, performed bacteriologic investigations of the flora of the external ear and demonstrated that a high percentage of these cases showed a Gram-negative bacillus, predominantly the *Pseudomonas aeruginosa.*

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Cultures were taken on the first visit, at three-day intervals thereafter, and on the day of discharge. Small, cotton-tipped applicators were employed for taking of cultures from both external auditory canals of every new patient. The material for culture was collected by gently inserting the tip into the ear canal. The swab was placed at once into thioglycollate broth. If the person was suspected of having otomycosis, another culture was sent for mycologic investigation. All cultures were held at room temperature until taken to the laboratory at the close of the afternoon clinic. The bacteriologic techniques have been described elsewhere.[5]

Sensitivity tests were performed routinely on the initial culture and thereafter whenever indicated. Tests were performed for dihydrostreptomycin, oxytetracycline, sulfadiazine, 4-aminomethylbenzenesulfonamide, and polymyxin. The results were recorded as very sensitive, moderately sensitive, or resistant. Nitrofurazone discs were not available and, therefore, no tests were performed with this preparation.

A total of 381 patients and 493 ears with acute diffuse external otitis were observed. Five series of cases were treated by the local application of antimicrobial agents and one with control preparations. These antimicrobial formulations have received wide usage and numerous scientific reports attest to their clinical effectiveness:[6,8,9,10,11,12]

1. Oxytetracycline HCl.[*]
2. Polymyxin B Sulfate.[†]
3. 4-aminomethylbenzenesulfonamide HCl 5 per cent.[††]
4. Nitrofurazone.[§]

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[*] Terramycin Otic. Oxytetracycline Hydrochloride 5 mg. per cc. Chas. Pfizer & Co., Inc., New York, New York.

[†] Polymyxin B (Sulfate) 200,000 Units equivalent to 20 mg. polymyxin standard for topical use only. (Dissolved in an isotonic saline vehicle to form a 0.1% solution.) Burroughs Wellcome & Co., Tuckahoe, N. Y.

[††] Sulfamylon Hydrochloride Viscous Solution 5%. Contains Sulfamylon (brand of 4-aminomethylbenzenesulfonamide) hydrochloride 5%; methylcellulose 1%; glycerine 8%; and, as preservative, methyl paraben 0.1%. Winthrop-Stearns, Inc., New York 18, N. Y.

[§] Furacin Anhydrous Ear Solution N.N.R. Brand of 0.2% Nitrofurazone. Contains 0.2% Nitrofurazone N.N.R. in a non-aqueous vehicle, polyethylene glycol 300. Eaton Laboratories, Norwich, New York.

5. Oxytetracycline HCl and Polymyxin B Sulfate.§§
6. Controls.
   a. Normal (0.85%) saline drops.
   b. Fatty acids in a lanolin vehicle.

To avoid any bias in the selection of antimicrobial therapy, the above medications were assigned in a pre-arranged order except where the extreme severity of the disease necessitated a combined local and systemic therapy. As patients were admitted to the ear clinic the recorder selected the next drug on the list for administration, and this choice was noted on the patient's chart; thus, the professional personnel had no control over the therapeutic agent given. Those cases receiving combined local and intramuscular antimicrobial therapy were not included in the series of cases studied comparatively.

In the clinical handling of these cases, it was neither practicable nor desirable to persist rigidly with an initial therapy to which the patient was not responding. If, after several days, the patient showed no improvement, the medication was changed. The new therapy was chosen on the basis of *in vitro* bacteriologic sensitivity reports; however, following a change of treatment there was uncertainty whether improvement was a delayed response to the original medication, to an early response to the new medication, or to combined action. Thus, in the evaluation of response to local therapy, the data included in this series are the observations on the initial therapeutic regime.

The dosage of the medications used in all cases was that suggested by the pharmaceutical companies. Drops were applied directly into the external auditory canal or by cotton wick moistened with the therapeutic agent. The technique selected was determined by the amount of edema of the skin of the ear canal. If the edema was rated three or more, the wick method was arbitrarily chosen. As soon as the edema subsided, drops were substituted and the wick eliminated. When a wick was used, five drops of drug were applied every four hours while the patient was awake. When the drops alone were used, the patient was instructed to lie on the unaffected side, place five drops in the involved ear, and remain in that position for five minutes. He was then asked to sit erect and allow the excess medication to drain out upon cotton held at the meatus.

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§§ Terramycin Otic. Oxytetracycline Hydrochloride 5 mg.; *Polymyxin B Sulfate* 10,000 Units; Benzocaine 5%; 90% Propylene Glycol per cc. Chas. Pfizer & Co., Inc., New York, New York.

Patients were requested to return daily except on weekends. Only severe cases were seen on Saturday, and the Clinic was closed on Sunday. Not uncommonly, patients did not return after making favorable progress, since follow-up visits were not required by military order. For purpose of tabulation, these patients were counted as cured as of the date of the last visit to the Clinic. Despite the admitted limitations of the statistical data due to such influences, it is to be noted that military personnel returned for observation with a regularity which is rarely attained in civilian practice.

From the detailed clinical data recorded on admission and on each follow-up visit, selected observations were used as indices of clinical change. These included the subjective manifestations of pain and tenderness and the objective finding of edema. The criteria for discharging a patient consisted of absence of pain, no edema of the external canal, and 2+ or less erythema. (A rating of 2+ or less erythema on discharge was permitted since the medication often caused a slight erythema.) A negative bacteriologic culture was not considered necessary for discharge since some percentage of patients continued to have Gram-negative bacilli in their external auditory canals after all symptoms and physical findings had disappeared.

RESULTS.

A total of 381 patients and 493 ears with acute diffuse otitis externa were observed. Two-hundred-and-sixty-nine were unilateral and 112 bilateral. The statistical data on follow-up treatment and examinations were satisfactory in 403. Excluding the 17 severe cases given both systemic and local antimicrobial therapy on the initial visit, there were 386 infected ears for comparative study of the effectiveness of the various therapeutic agents and procedures used. The local medications employed and the number of cases with follow-up data for each treatment series is shown in Table 1.

The comparability of the series of cases under observation has been examined in varying ways. The data in Table 2 are

TABLE 1.

LOCAL MEDICATION EMPLOYED AND THE NUMBER OF CASES OBSERVED WHERE THERE WERE ADEQUATE FOLLOW-UP EXAMINATIONS.

Series* No.	*Local Medication*	Number of Cases
1	Terramycin otic	67
2	Polymyxin in saline	95
3	Sulfamylon aqueous	77
4	Furacin otic	65
5	Terramycin-Polymyxin otic	50
6	No anti-microbial therapy	32
	TOTAL	386

* In subsequent tables, therapy employed is shown by treatment series number.

TABLE 2.

REPRESENTATIVE DATA INDICATING THE COMPARABILITY OF CASES IN THE DIFFERENT TREATMENT SERIES.

Characteristic	Percentage of all cases with the indicated characteristics by treatment series.*						
	Average	1	2	3	4	5	6
Severity — Mild	50	42	49	53	48	50	63
No previous otitis externa	50	57	50	53	47	42	48
Age — Under 25 years	64	64	61	71	71	52	63
Wax in ear, excessive	32	36	24	31	33	43	33
Pain — Moderate to severe	62	61	61	70	64	55	70
Itchiness	38	38	41	30	36	18	36
Feeling of Fullness in Ear	75	80	77	71	73	75	70
Initial culture positive for *Pseudomonas*	76	72	76	86	78	58	87

* See Table 1.

illustrative. The severity of the disease in each of the series was closely similar except for Series 6. These cases tended to be of the milder category. It is possible that the more severe cases were rejected as proper subjects for the use of saline drops or fatty acid preparations only. The degree of pain in the different treatment series was closely similar.

Series 5 differs somewhat from the others. This may be explained in part by the fact that this series was initiated in the latter part of the study as a substitute for one of the original therapeutic agents, the manufacture of which was discontinued. At that time, there were fewer students at the Center. With a reduction of young trainees, a higher proportion of the cases were regular Navy personnel of older age. Also a lower percentage of Series 5 was positive for *Pseudomonas*. This is in line with findings of 1952 when the percentage of positive cases declined beginning in September.[13] The total score rating for the severity of pain and tenderness and the rating for the degree of edema are given in Tables 3 and 4. In considering these, it is to be remembered that not all patients returned daily, and in general those progressing least favorably were the ones seen most frequently. These distorting influences become more marked by days of therapy and the observations presented here are limited, therefore, to five days from the beginning of treatment.

The data in Tables 3 and 4 show the progress of all patients to be relatively slow. When measured by the subjective symptoms of pain and tenderness, or the objective finding of edema, Series, 1, 2, 3, and 5 progressed more rapidly than those in Series 4 or 6. Treatments 1 and 5 seem to have given the greatest amount of relief from pain and tenderness and a somewhat more rapid response to therapy. Series 5 showed almost complete resolution of pain and tenderness on the fourth day. Progress was less favorable with treatments 2, 3, and 4 in this category. No appreciable difference appeared in the rate of decline of pain and tenderness in patients on treatment 4 as compared with those receiving no antimicrobial therapy on the fourth and fifth days.

As previously mentioned, it was believed that the edema of the ear canal provided a more objective measure of change in

TABLE 3.

THE AVERAGE "SEVERITY SCORE" FOR PAIN AND TENDERNESS BY DAY OF TREATMENT AND THERAPY EMPLOYED.*

Day of Treatment	Average "Severity Score" by Treatment†						Average "Severity Score" by Treatment As a Per Cent of the Initial Average Score					
	1	2	3	4	5	6 & 7	1	2	3	4	5	6 & 7
Initial	7.84	7.51	8.56	8.87	5.46	7.85	100.0	100.0	100.0	100.0	100.0	100.0
1	6.28	6.81	6.31	7.00	5.30	5.96	80.1	90.7	73.5	78.9	97.1	75.9
2	4.12	3.18	3.61	4.51	2.42	4.83	52.6	42.3	42.2	50.8	44.3	61.5
3	2.41	3.11	3.60	2.78	3.00	4.92	30.7	41.4	42.1	31.3	54.9	62.7
4	1.27	1.33	2.01	2.11	0.11	1.86	16.2	17.7	33.8	23.8	2.0	23.7
5	0.50	1.31	1.00	2.44	0	2.17	6.4	17.4	11.7	27.5	0	27.6

* See Table 1.
† Maximum Score, pain 10, tenderness 10, Total 20.

TABLE 4.

THE AVERAGE "SEVERITY SCORE" FOR EDEMA BY DAY OF TREATMENT AND THERAPY EMPLOYED.*

Day of Treatment	Average "Severity Score" by Treatment†						Average "Severity Score" by Treatment As a Per Cent of the Initial Average Score					
	1	2	3	4	5	6 & 7	1	2	3	4	5	6 & 7
Initial	3.18	3.18	2.87	2.60	2.70	2.38	100.0	100.0	100.0	100.0	100.0	100.0
1	3.02	2.82	2.42	2.61	2.95	2.23	95.0	88.7	84.3	100.4	109.3	93.7
2	2.49	2.15	1.59	1.97	2.10	2.17	78.3	67.6	55.4	75.8	77.8	91.2
3	1.63	1.58	1.66	1.83	2.18	1.83	51.3	49.7	57.8	70.4	80.7	76.9
4	0.95	1.38	0.91	1.50	1.22	1.86	29.9	43.4	31.7	57.7	45.2	78.2
5	0.25	1.19	0.93	1.56	0.75	1.50	7.9	37.4	32.4	60.0	27.8	63.0

* See Table 1.

† Maximum score 10.

TABLE 5.

NUMBER AND PER CENT OF PATIENTS WHOSE THERAPY WAS CHANGED BY DAY OF TREATMENT AND INITIAL THERAPY EMPLOYED.*

Day of Treatment	Cumulative Number of Patients Switched at End of Each Day of Treatment						Cumulative Number of Patients Switched As a Per Cent of Total Patients					
	1	2	3	4	5	6 & 7	1	2	3	4	5	6 & 7
Total Patients	67	95	77	65	50	32	100.0	100.0	100.0	100.0	100.0	100.0
1	1	10	7	1	2	8	1.5	10.5	9.1	1.5	4.0	25.0
2	3	11	14	3	5	9	4.5	11.6	18.2	4.6	10.0	28.1
3	7	14	16	6	6	13	10.4	14.7	20.8	9.2	12.0	40.6
4	10	15	17	6	7	17	14.9	15.8	22.1	9.2	14.0	53.1
5	10	19	18	7	7	17	14.9	20.0	23.4	10.8	14.0	53.1
End of Treatment	11	26	19	11	7	19	16.4	27.4	24.7	16.9	14.0	59.4

* See Table 1.

TABLE 6.

NUMBER AND PER CENT OF PATIENTS "CURED" BY DAY OF TREATMENT AND THERAPY EMPLOYED.*

Day of Treatment	Cumulative Number of Patients "Cured" at End of Each Day of Treatment						Cumulative Number of Patients "Cured" As a Per Cent of Total Patients					
	1	2	3	4	5	6 & 7	1	2	3	4	5	6 & 7
Total Patients	67	95	77	65	50	32	100.0	100.0	100.0	100.0	100.0	100.0
1	6	11	4	10	4	0	9.0	11.6	5.2	15.4	8.0	0
2	9	20	11	23	15	1	13.4	21.1	14.3	35.4	30.0	3.1
3	20	25	19	30	23	2	29.9	26.3	24.7	46.2	46.0	6.2
4	30	37	30	39	27	3	44.8	38.9	39.0	60.0	54.0	9.4
5	33	42	35	43	30	4	49.3	44.2	45.5	66.2	60.0	12.5
End of Treatment	56	69	58	54	43	13	83.6	72.6	75.3	83.1	86.0	40.6

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* See Table 1.

the condition of the ear canal. It is seen that Table 4 confirms the previous observation on pain and tenderness. Series 1 and 5 show the most rapid reduction in edema and the greatest total improvement after five days. Series 4 offers very little more improvement than seen with the control group. On the other hand, Series 2, 3, and 5 show very similar response to therapy.

Further comparative data are given in Tables 5 and 6. The number and percentage of cases in which the initial therapy was considered ineffective are given in Table 5. Here the wide difference between those receiving and those not receiving antimicrobial therapy is evident. The number of ears in which therapy was changed was less in Series 1 and 5 than in Series 2 and 3. The related findings in Table 6 showing the number and percentage of ears cured by the initial therapy tend to support the opinion that treatments 1 and 5 were superior to 2 and 3.

There was, however, a sharp divergence in the findings for treatment 4. The data in Tables 3 and 4 suggest a poor response of pain and tenderness and edema while Tables 5 and 6 indicate that this form of therapy is equal in effectiveness to the other preparations in the number of ears cured and in the number requiring a change in medication.

Of some concern in the use of the preparation in Series 4 was the observation for possible local sensitivity reactions. Minor local reactions could reasonably account for the less favorable rating of this preparation when measured by pain, tenderness, and edema; however, no such reactions necessitating a change of drug were observed in this series.

Although follow-up bacteriologic examinations were limited since repeat cultures were taken ordinarily only every third day of treatment, the striking feature of the findings as shown in Table 7 is the persistence of Pseudomonas in the ear canal. These data, however, fail to indicate the quantitative change in the relative numbers of Pseudomonas present in the ear. The major change observed during treatment was the decline in the number of Pseudomonas colonies on positive plates. In 126 cases, terminal cultures were taken as the patient was being discharged as cured and even here 25 per cent were positive for Pseudomonas. A smaller number of ears showed positive terminal cultures in Series 5 than in the others (See Table 7).

TABLE 7.

THE PERCENTAGE OF CASES WITH CULTURES POSITIVE FOR *Pseudomonas aeruginosa* BY DAY OF TREATMENT AND THERAPY EMPLOYED.*

Day of Treatment	Therapy*					
	1	2	3	4	5	6
Initial	72	76	86	78	58	87
1 – 2	50	69	87	78	22	84
3 – 4	52	44	90	53	21	85
5 – 7	75	50	75	56	31	84

* See Table 1.

Analysis of the data on the 17 severe cases treated initially by systemic and local antimicrobial therapy indicated that pain and tenderness began to decrease gradually after 24 hours, but that there was no substantial reduction in erythema or edema for the first four days of treatment. The limited bacteriologic data indicated that the combined systemic and local medication was no more effective than the Series 5 preparation alone in controlling the growth of bacteria obtained on culture of the ear canal.

Results of *in vitro* sensitivity tests performed on Pseudomonas isolated on initial cultures are shown in Table 8. These observations suggest that a higher percentage of the cultures are sensitive to oxytetracycline HCl and sulfadiazine. Of all Pseudomonas examined *in vitro* for sensitivity to oxytetracycline, 4 per cent were found to be resistant. Of 66 found resistant to polymyxin, and also tested for sensitivity to oxytetracycline, only three (4 per cent) were also resistant to this compound. There was no suggestion of any correlation in the sensitivity or resistance of Pseudomonas to these two compounds.

TABLE 8.

SENSITIVITY OF *Pseudomonas* TO VARIOUS ANTIBIOTICS ON INITIAL EXAMINATION.

Antibiotic	Cultures					Per Cent Distribution				
	Total Tested	Resistant	Moderately Sensitive	Very Sensitive	Sensitive Degree Not Stated	Total Tested	Resistant	Moderately Sensitive	Very Sensitive	Sensitive Degree Not Stated
Terramycin	281	11	142	128	—	100.0	3.9	50.5	45.6	—
Streptomycin	281	241	33	7	—	100.0	85.8	11.7	2.5	—
Polymyxin	216	66	—	—	150	100.0	30.6	—	—	69.4
Sulfadiazine	281	14	24	243	—	100.0	5.0	8.5	86.5	—
Sulfamylon	281	20	115	146	—	100.0	7.1	40.9	52.0	—

DISCUSSION.

The preliminary observations in the evaluation of therapy of otitis externa in 1952 in Florida were in line with the findings of the more comprehensive study of 1953. The conclusion of the preceding year that "no therapeutic agent could be considered to be uniformly and highly satisfactory" is not challenged by the more recent findings. The response to therapy in many cases was "discouragingly slow."

It is interesting to note that remarkable results were obtained with each of the drugs in certain instances, even when fatty acids or saline preparations alone were used. Thus the general impression was confirmed that thorough cleansing and prophylactic care of mild cases often produced results without recourse to antimicrobial medication. This was not true of the more severe varieties which often necessitated more than one therapeutic agent or procedure to control the disease. In only a few instances were the reactions so severe that hospitalization was recommended. It is believed that further efforts utilizing a larger series should be made to study the response of the various grades of disease to treatment.

It should be emphasized that the unique opportunity of studying a large series of cases under almost ideal conditions at a military base provided unusual control of patients as well as an opportunity to select only cases of acute diffuse external otitis for this study. All patients with complicating generalized dermatologic problems, seborrheic dermatitis, or localized or disseminated neurodermatitis, were rejected for use in this investigation. A clear distinction between furunculosis and diffuse external otitis was made, and wherever there was any question, follow-up cultures and clinical observations were made before it was decided to include the patient in this series.

The practical importance of *in vitro* sensitivity tests is not fully established. At best these should serve only as a guide for therapy; they cannot infallibly predict the effectiveness of any given medication when applied locally. Where a choice exists as to a medication to be applied topically, it is natural that the choice should go to the preparation which has the greatest *in vitro* activity; however, other factors such as effectiveness in the presence of exudates and secretions modify the therapeutic response, and these factors must be weighed by the otologist in the selection of the medication of choice.

A limitation in the clinical importance of bacteriologic cultures deserves comment. It is the opinion of one of us (B.H.S.) that the Pseudomonas infection is a superimposed disease and that the primary disease exists deep in the dermis and must be approached in some manner other than by local antimicrobial therapy. This theory does not minimize the necessity of efficacious local surface therapy to eradicate the pseudomonas organisms. It is believed, however, that further investigation will provide the evidence which will show that the proper approach to therapy of the more severe varieties of acute diffuse external otitis is by way of the parenteral route, thus attacking directly the deeper inflammation present in and around the apocrine glands.

SUMMARY.

1. A total of 493 ears with acute diffuse external otitis was observed, 269 unilateral and 112 bilateral. Of the 493 cases, 403 returned for follow-up treatment and examination; 17 were given combined systemic and local therapy, thus leaving 386 infected ears in the series studied comparatively.

2. These 386 were divided into 6 series and were treated with the following preparations:—

  1) Oxytetracycline HCl (containing 5 mg. per cc.).

  2) Polymyxin B Sulfate (dissolved in an isotonic saline vehicle to form a 0.1 per cent solution).

  3) 4-Aminomethylbenzenesulfonamide HCl (commercial 5 per cent aqueous solution).

  4) Nitrofurazone Anhydrous Ear Solution (containing 0.2 per cent Nitrofurazone in a non-aqueous vehicle).

5) Oxytetracycline-Polymyxin Otic (contains 5 mg. of oxytetracycline and 10,000 Units of polymyxin per cc.).

6) Controls.

  a) Normal (0.85 per cent) saline drops.

  b) Fatty acids in a lanolin vehicle.

3. None of the therapeutic agents studied could be considered to be uniformly and highly satisfactory. In some cases response to therapy was discouragingly slow.

4. When measured by subjective symptoms of pain and tenderness and the objective finding of edema, oxytetracycline, or a combination of oxytetracycline and polymyxin, seemed to offer slightly better results than other preparations.

5. When the number of patients cured after five days as well as the number of patients requiring an alteration of therapy are compared, there is little difference in the effectiveness of the various antimicrobial agents.

6. Bacteriologic observations confirm the high incidence of Pseudomonas in acute diffuse external otitis. The major change observed during treatment was the decline from many to few Pseudomonas found in cultures. In 25 per cent of the patients reported clinically cured, Pseudomonas could still be cultured from the external ear canal.

7. *In vitro* sensitivity tests performed on Pseudomonas isolated on initial cultures revealed that the least number of strains were resistant to sulfadiazine and oxytetracycline. Thus, only 4 per cent of the strains found resistant to polymyxin and also tested for sensitivity to oxytetracycline, only 3 per cent were resistant to this compound.

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BRONCHOESOPHAGOLOGY COURSE.

The next Bronchoesophagology Course to be given by the Temple University School of Medicine is scheduled for January 31st - February 11th, 1955, under the direction of Dr. Chevalier L. Jackson.

Interested registrants will please write directly to the Department of Laryngology and Bronchoesophagology, Temple University School of Medicine, 3400 North Broad Street, Philadelphia 40, Pa.

BACTERIOLOGICAL STUDIES OF OTITIS EXTERNA

1951, 1952, AND 1953.*

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Singer, *et al.*, in 1951, examined bacteriologically and mycologically 1,377 normal and 646 infected external auditory canals.[1] They concluded, as have other authors, that the mycological flora had limited, if any, etiological significance. The *Pseudomonas* group of organisms appeared to have an important etiological role though other Gram negative bacilli and streptococci also occurred more commonly in the infected than in the normal ears. The study upon which these tentative conclusions were based was primarily a laboratory investigation. The individuals examined were contacted only when cultures were taken, and there was no correlation of clinical data with the laboratory findings. During the two following years, these bacteriological investigations have been continued in association with clinical studies of the relative efficacy of anti-microbial therapy. This report comprises a summary of all findings in this laboratory on the bacteriological flora of the normal ear and those with otitis externa.

The individuals studied during these investigations were young adult males serving either at the U. S. Naval Air Sta-

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tion in Jackson da, or at McDill Field i Florida. During 19. study was limited to Jac Clinical dat on the presented separately by et al.[2] In year vestigations were conducte the summ nths ning in June and termin October. During 1952 1953 at the Naval Air Stat in 1952 also at McDill F d a special "Ear Clinic" wa lished. All service pers el applying for treatment of disord , possibly mably due to otitis extern referr to the Clin diagnosis and treatment. A numbe of civilian dents were seen also. On all seen i 952 and 19 iled clinical observations we lected nd were avai or study.

Th culture proced mployed were essentially the throughout the entir y. The media were prepared dehydrated products) according to the manufactu recommendation. Th sed included fluid thioglycollate dium (in 16 x 125 mm bes), eosin-methylene blue agar, bloo agar (Bacto p s No. 3 agar plus 5 per cent w citra d human blood. Cultures were taken by one of participating bacteriologists or by the physician at the cli Steri , small, cotton-tipped swabs were used, and mater for culture was obtained by gently inserting the tip into t canal of the infected or normal ear. In 1952, all cultures were taken n duplicate. One was used for a direct streaking of an eosin-methylene blue plate, all other swabs were placed immediatel into a tube of thioglycollate broth. This, and the inoculated eosin-methylene blue plates were taken to the laboratory a the conclusion of the afternoon clinic and were incubated overnight at 37° C. The broth, if sufficiently turbid to indicat growth, was examined after this period of incubation. Those remaining clear were incubated for 18 hours before being discarded as negative. Transplants from the broth were p ated to both blood agar and EMB agar. Standard techniques were employed for the isolation and the identification of organisms.

The bacteriological findings in cases of otitis externa and in comparable cultures taken from the normal ear are sh n Table 1. The normals examined in 1953 were service p nel observed in a trial of prophylaxis of otitis externa

TABLE 1.

BACTERIOLOGICAL FINDINGS IN CULTURES OF NORMAL AND INFECTED EXTERNAL AUDITORY CANALS 1951, 1952 AND 1953.

Organism	Number and Per Cent of Cultures for Specified Organism									
	Acute Otitis Externa						Normal Ears			
	1951		1952		1953		1951		1953	
	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
Pseudomonas Group	423	65.5	375	80.8	371	75.5	14	1.0	29	1.3
Proteus Group	53	7.9	45	9.7	41	8.3	5	.4	5	.2
Coli-Aerogenes Group	213	32.9	152	32.7	111	22.6	72	5.2	90	4.0
Alcaligenes Group	43	6.7	20	4.3	18	3.6	22	1.6	24	1.1
Paracolon Group	6	.9	11	2.4	6	1.2	5	0.4	2	.1
Alpha-type Streptococci	22	3.4	25	5.4	10	2.0	24	1.7	4	.2
Beta-type Streptococci	25	3.9	10	2.2	20	4.0	0	0	2	.1
Gamma-type Streptococci	49	7.6	65	14.0	42	8.5	15	1.1	515	23.4
Beta-hemolytic Staphylococcus Albus	174	26.8	131	28.2	171	36.6	801	58.8	1,420	64.5
Non-hemolytic Staphylococcus Albus	81	12.5	129	27.8	49	9.8	467	32.5	1,065	48.4
Beta-hemolytic Staphylococcus Aureus	64	9.9	70	15.0	42	8.5	128	9.3	24	1.1
Non-hemolytic Staphylococcus Aureus	40	6.1	45	9.7	2	.4	90	6.5	33	1.5
Beta-hemolytic Staphylococcus Citreus	3	.4	2	.4	2	.4	19	1.4	14	.6
Non-hemolytic Staphylococcus Citreus	1	.1	0	0	0	0	15	1.1	26	1.2
Chromogenic Soil Organisms	4	.6	2	.4	5	1.0	34	2.5	0	0
Spore-formers only	0	0	26	5.6	1	.2	182	13.2	18	.8
Diphtheroids	Omitted	–	10	2.2	17	3.4	Omitted	–	374	17.0
Total Examinations	646		464		491		1,377		2,200	

results reported by Senturia.[3] With few exceptions, there is a high degree of consistency in the observations on cases of acute otitis externa in each of the three years and on the normal ears during the two years this examination was performed. The variations in the percentage of normal ears with Gamma type streptococci and of Beta-hemolytic and Non-hemolytic staphylococcus aureus in 1951 and 1953 cannot be securely explained but presumably indicate a true difference in the distribution of this flora.

The proportion of cases of acute otitis externa found positive for organisms of the *Pseudomonas* group varied in part with the criteria employed in diagnosing this infection. In 1952 the cases referred to the "Ear Clinic" were divided into four groups as reported by Yawn, *et al.*[4] Acute cases with pain, diffuse edema, redness of the canal and a definite purulent exudate were accepted as acute otitis externa and 80.8 per cent of these were positive for *Pseudomonas.* A small number had had symptoms for over 30 days prior to treatment. These were classified as chronic otitis externa and 37.5 per cent of these were positive for *Pseudomonas.* There were also some very mild disorders with itching, or slight pain, little if any redness and no purulent exudate. Lacking an expert opinion, these were not finally diagnosed. Only 24.3 per cent of these were positive for *Pseudomonas.* At McDill Field in 1952 the cases referred to the "Ear Clinic" were typical and relatively severe and of these 90.5 per cent yielded *Pseudomonas.* In Jacksonville during 1952, 84.0 per cent of those considered to be severe cases were positive as compared with 71 per cent in those of moderate severity. The comparable percentages for 1953 were 85.5 and 75.1 respectively.

The consistent rarity of *Pseudomonas* in the normal external ear canal as compared with its occurrence in large numbers in a high percentage of the exudative cases of otitis externa was striking. The conclusion that it is implicated as the sole or an associated cause in a very high proportion of these cases of otitis externa appears warranted.

The other Gram negative bacilli also were found more commonly in the infected as compared with the normal ear. Members of the *Proteus* group were found with particular frequence in cases which proved resistant to treatment. The bacteriological evidence suggested that these and the Alpha and Beta Type Streptococci may have a less important and less frequent role in the etiology of otitis externa. There was no evidence to implicate other bacteria. While extensive mycological examinations were conducted by us only in 1951, still the clinical observations in 1952 and 1953 did not provide evidence to question the conclusion that mycological flora play a very limited role in the etiology of acute otitis externa.

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TWO HUNDRED BRONCHOSCOPIES IN PULMONARY TUBERCULOSIS.*

A Comparison with Corresponding Findings in the Pre-streptomycin Era.

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Many years passed after the discovery of bronchoscopy, before this method was employed systematically in the diagnosis and therapy of tracheo-bronchial tuberculosis. For a considerable time bronchoscopy was judged to be a dangerous *procedure in pulmonary tuberculosis.*

Von Eicken,[1] the pupil and successor of Killian as the chief of the Department of Otolaryngology at the University of Berlin, published a survey of the history of bronchoscopy in 1939, and even then did not mention the application of this procedure in pulmonary tuberculosis.

Myerson,[2] in 1928, was the first clinician to employ bronchoscopy in this disease. The 1930s were the pioneer years in the use of bronchoscopy on tuberculous patients, by Myerson, Kernan,[3] and Clerf.[4] Due largely to their efforts, bronchoscopy is today used as a very important, one might almost say indispensable, aid in the diagnosis of bronchial tuberculosis. Except for severe tuberculosis of the larynx and a poor general condition, there are few contraindications to bronchoscopy in pulmonary tuberculosis.

The severity and incidence of pulmonary tuberculosis and its complications has been considerably reduced by the treatment with streptomycin and other antibiotics. To prove that the same happened in bronchial tuberculosis one has only to

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study the pertinent literature of the years prior to 1946. Following is the essence of literature from 1936 to 1944, published in a review by Holinger.[5]

The incidence in patients, in whom a bronchial involvement was *suspected*, was *30 to 60 per cent, while perforations* of tuberculous lymph nodes into the trachea or bronchi occurred in only 1.3 per cent.

Four pathological groups of bronchial tuberculosis must be differentiated: *1.* Submucosal involvement; *2.* Ulcerations with granulations; *3.* Hyperplasia; *4.* Stage of healing or fibrosis.

The route of infection is either direct surface implantation, direct extension from the parenchymal lesion, or from the hilar nodes. Rarely occurs a lymphogenous or hematogenous spread. Incidence of bronchial obstruction was high.

The literature in this field is quite extensive, and I quote but a few authors.

Froste[6] performed 1000 bronchoscopies on 420 patients with pulmonary tuberculosis between 1944 and 1947. His findings were specific tuberculous bronchitis, atelectasis, bronchostenosis, lymphadenitis with perforation into the bronchus, and bronchogenic cancer. There was a combined frequency of 37.2 per cent of positive findings, while the incidence of perforated lymph nodes into the bronchus was only 1.2 per cent.

Acuna's[7] statistics showed 29.7 per cent positive cases of bronchial tuberculosis. Besides the usual lesions he saw displacement of trachea and bronchi, deformities of the lumen, and partial, or total stenosis of the bronchi.

Besides other indications for bronchoscopy, mentioned by Acuna, we consider positive gastric cultures which are not consistent with the clinical and roentgenological findings and also certain conspicuous markings of the bronchi on the X-ray picture as another indication for bronchoscopy.

An unusually high percentage of bronchial tuberculosis was found by Stone[8] in 1945, at the Norfolk County Sanatorium, Mass. Of all patients bronchoscoped because of suspicious symptoms, 50 per cent showed evidence of endobronchial tuberculosis.

Myerson[9] in 580 patients, bronchoscoped before 1941, found 160 patients with evidence of endobronchial disease, 28 per cent. All lesions with the exception of granulomata secondary to perforating mediastinal glands, were due to an ulcerous lesion in the lung parenchyma. Myerson points out that tuberculosis of trachea and bronchi follows a definite course, in which the various predictable stages can be distinguished.

In most instances the granuloma, extending from the branch bronchus, fills the main bronchus with a resultant atelectasis of the entire lung. As soon as the granuloma occludes the bronchus, the sputum becomes negative.

Tuberculosis of the finer bronchi and bronchioles cannot be proved, but it occurs in a very high percentage. Myerson thought that the treatment of choice at this time, namely cauterization with 30 per cent silver nitrate solution and the dilatation of a stenosed bronchus, rarely helped the patient, and sometimes probably harmed him. Lloyd[10] expressed the same opinion in a study based on 475 bronchoscopies, the most of which were also performed at Sanatorium Otisville. He saw no cases in which cauterization had been of any value and observed no permanent increase of the lumen of a bronchus following dilatation.

Hemoptysis, reappearance of tubercle bacilli, and ulcers have followed the instrumental stretching of a healthy scar. The frequency of positive findings, however, was so high, that abnormal findings were present in practically all cases. A number of mechanical changes were observed by this author like torsions and distortions, clockwise and counterclockwise rotation of bronchi and carina, and inward bulging of bronchi, probably due to pressure of mediastinal lymph glands. He warns against collapse therapy in certain cases of bronchial tuberculosis, since it could become a serious impediment to pulmonary drainage and thus might lead to secondary atelectasis.

Pathological findings in autopsies showed even a higher percentage of bronchial tuberculosis than bronchoscopy.

Auerbach[11] in 1949 published a study of bronchial lesions obtained in 1000 autopsies of tuberculous patients. There was evidence of tracheo-bronchial tuberculosis in 421 cases, *i.e.* 42.1 per cent. In only 12 cases, *i.e.* 1.2 per cent was the lesion due to a rupture of tuberculous regional lymph glands into the tracheo-bronchial tree.

In another post mortem study, based on 125 autopsies and performed before 1943, Salkin,[12] *et al.*, observed about the same percentage of bronchial tuberculosis as Auerbach, namely 40 to 50 per cent. Auerbach's and Salkin's post mortem findings prove that in most cases of cavitation the draining bronchus is involved.

Today, under treatment with streptomycin, PAS, isoniacid, collapse therapy, and surgery, most of these lesions will heal without producing symptoms.

When the pulmonary lesion improves it often heals spontaneously even before streptomycin and other methods of treatment are applied.

A conspicuous difference exists in the occurrence of perforations of hilar lymph glands into trachea and bronchi as reported in the United States of America and Europe. According to the reports of Froste[6] and Auerbach[11] the incidence of this complication in the United States is about 1.2 per cent, Holinger quotes 1.3 per cent; Schwarz,[13] in Europe, on the other hand, reports about 106 scars due to such perforations among 426 autopsies, or about 25 per cent. Hanns[14] confirms the latter's report and is convinced that the bronchial tuberculosis secondary to perforation of caseous hilus glands has widely increased in the decade between 1938 and 1948. Dufourt[15] and Monnier made the same observation.

This conspicuous difference in the incidence of perforation of caseous hilus glands into the tracheo-bronchial tree between the reports from Europe and the United States may be related to the malnutrition of large sections of the population in many European countries during the war and postwar years. Such nutritional deficiencies probably hastened the necrotic process in the mediastinal glands, so that there was no time for the erection of a protecting wall around the glands by fibrotic thickening of the capsule. This type of bronchial tuberculosis occurs not uncommonly without specific involvement of the lungs; however, stenosis and atelactasis are frequent complications.

Brunner[16] and Soulas[17] warn against collapse therapy in bronchial stenosis because of the danger of complete and irreversible atelectasis. Tuttle,[18] *et al.*, consider bronchial stenosis a very serious complication. Of their 92 patients with bronchial stenosis, 23 patients (32 per cent) died of poststenotic suppuration and extension of the bronchial disease, and they conclude that patients with healed stenosis face great uncertainty. The incidence in empyema and unexpanded lungs in cases of bronchial stenosis is as high as 39.9 to 46.7 per cent. His fatality rate of 27 patients with poststenotic suppurations was 55 per cent.

The postbronchoscopic state is not always uneventful. Radner[19] observed a number of incidents among 183 bronchoscopies. In 18 cases a postbronchoscopic rise of temperature occurred within 72 hours. This was a nonspecific reaction without any significance. In 26 cases a secondary rise of temperature between the tenth and sixteenth day occurred following bronchoscopy. Many of these patients complained of headache, anorexia, and increased cough. The febrile period usually lasted from two to three weeks, but extended sometimes into months, accompanied by manifest changes in the X-ray pictures. Radner quotes several other authors who saw a spread of the disease following bronchoscopy. He is of the opinion that this secondary rise of temperature may be due to the formation of a new, or the reactivation of an old focus.

Salkin,[13] *et al.*, noted a fair number of reactions, such as nausea, vomiting, dizziness, and even syncope, due to the local anesthetic of 2 per cent pontocaine solution.

At Otisville all bronchoscopies are performed under local anesthesia with 10 per cent cocaine solution for the larynx and a 3 per cent solution for the bronchi. The premedication consists of nembutal 1.5 gr. on the evening prior to bronchoscopy. This is repeated in the morning and 1/6 gr. morphine with 1/150 gr. atropine in 5 cc. normal saline solution is administered intravenously immediately before the local anesthesia is started. Except for some dizziness after the morphine injection which passes within a few minutes, we rarely had unpleasant reactions. It is our impression that the cocaine solution is less toxic than the pontocaine solution due to our premedication. There is evidence that the barbiturates reduce the toxic effect of cocaine. Bastedo[20] recommends barbiturates for the prophylaxis and treatment of cocaine poisoning.

Our own material consists of 200 bronchoscopies, which were performed on 188 patients at the Municipal Sanatorium, Otisville, N. Y., in 1951 and 1952. All these bronchoscopies were performed by our visiting physicians, the late Dr. Lloyd, Dr. Chochet, of New York City, and Dr. Feldman, of Middletown, N. Y. The anesthesia was done by the author.

Our findings follow:

1. Partial or complete obstruction of the main bronchus or one of its branches, buckling and bulging of bronchi, 11 cases, 6 per cent;

2. Clockwise or counterclockwise rotation of carina of a main bronchus or its branches, 11 cases, 6 per cent;

3. Edema of the mucous membrane of the bronchus, 10 cases, 5.3 per cent;

4. Granulations in the lumen of the bronchi, 10 cases, 5.3 per cent.

5. Inflammatory acute infiltrative reaction, three cases, 1.6 per cent;

6. Bulging of trachea or main bronchus due to enlarged mediastinal glands, three cases, 1.6 per cent;

7. Granulomata, two cases, 1.1 per cent;

8. Ulcerations, two cases, 1.1 per cent;

9. Atelectasis due to obstruction, two cases, 1.1 per cent.

Several of these conditions were often present in the same patient. Evidence of endobronchial tuberculosis was found in

29 patients, or 15 per cent. This incidence is significantly lower than the figures of 30 to 50 per cent, which were given by a number of other authors before the introduction of the treatment of pulmonary tuberculosis with antibiotics.

All of our cases of endobronchial tuberculosis occurred in patients with moderate or far advanced pulmonary tuberculosis. Not one case of perforation of enlarged caseous hilus glands into the bronchus was seen, although in three patients we found a bulging of the trachea or main bronchus, obviously due to pressure by enlarged mediastinal glands. We seldom observed a primary rise of temperature following bronchoscopy. In seven patients the temperature rose slightly on the first or second day after bronchoscopy and returned to normal within 24 hours. In one case a slightly increased temperature of 99 degrees persisted for ten days. We never saw a secondary rise of temperature with spread of the disease, which Radner[19] observed in 14.3 per cent of his cases.

The aspect of endobronchial tuberculosis today is completely different from that of the prestreptomycin period. Myerson[9] had already expressed the opinion, and Lloyd[10] concurred in it, that the treatment of endobronchial tuberculosis with 30 per cent silver nitrate solution, the standard procedure of that time — did more harm than good to the patient. Modern treatment employs streptomycin, PAS, isoniacid, collapse therapy, and surgery. Local treatment of the bronchi has become obsolete.

Although we frequently saw a narrowing of the bronchi due to edema, swelling of the mucous membrane, and granulations, we failed to see the fibrotic and scarred bronchi which, according to Tuttle[18] accounted for empyema, unexpanded lungs, and suppurations in 39 per cent with a high fatality. With the exception of six patients who are still in the Hospital improving, all other patients were discharged as inactive, with the working ability of four hours a day. The fibrotic scars which we do see, never cause a severe stenosis of the bronchus. We agree with Myerson and Lloyd that the treatment with silver nitrate solution contributed widely to the formation of fibrotic scars and stenosis of the bronchi. Only with 1/150 gr. atropine in 5 cc. normal saline solution is administered intravenously immediately before the local anesthesia is started. Except for some dizziness after the morphine injection which passes within a few minutes, we rarely had unpleasant reactions. It is our impression that the cocaine solution is less toxic than the pontocaine solution due to our premedication. There is evidence that the barbiturates reduce the toxic effect of cocaine. Bastedo[20] recommends barbiturates for the prophylaxis and treatment of cocaine poisoning.

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Some of the deformities, revealed by bronchoscopy in our case material, such as rotated carina, angulated bronchi, buckling and collapsed openings of bronchi, were probably caused by pneumoperitoneum. In one case inspissated mucous caused the obstruction of a main bronchus. A contracted right upper lobe was the cause of an acute angulation of the right upper lobe bronchus.

The conclusion can be reached that endobronchial tuberculosis today is one of the complications of pulmonary tuberculosis, the incidence and severity of which has been considerably reduced by modern methods of treatment.

SUMMARY.

For many years bronchoscopy has been considered a dangerous procedure in pulmonary tuberculosis; however, 20 years ago this procedure was introduced as the most important aid in the diagnosis of endobronchial tuberculosis.

The frequency of endobronchial tuberculosis was comparatively high before the introduction of streptomycin. A review of the literature has shown a reported frequency of 30 to 50 per cent positive findings in bronchoscopies and 40 to 50 per cent in autopsies. Granulations and ulcers, treated with 30 per cent silver nitrate solution, healed frequently with fibrotic scars, and stenosis of the main bronchus or one of its branches resulted. This was followed by atelectasis, insufficient expansion of the lung after pneumothorax, empyema, and suppurations of lung parenchyma with a high fatality rate.

The whole aspect of endobronchial tuberculosis improved with the introduction of streptomycin and PAS, and within the last two years of isoniacid. These drugs comprise the therapy of choice in pulmonary and endobronchial tuberculosis. The endobronchial tuberculosis usually heals without scar formation, and, therefore the incidence of bronchial stenosis and its sequelae is greatly reduced. In our material of 200 bronchoscopies the incidence of endobronchial tuberculosis has been 15 per cent as compared to an incidence of 30 per cent or more reported in the prestreptomycin era.

A number of deformities was revealed by bronchoscopy in our case material, as rotated carinae, angulated bronchi, buckling of bronchi, and collapsed openings of bronchi. Some of them were probably caused by pneumoperitoneum.

Complications due to local anesthetic were rare and never dangerous. We feel that the anesthesia with 10 per cent cocaine solution for the larynx and 3 per cent solution for the bronchi may produce fewer reactions than 2 per cent pontocaine solution, since it is possible to reduce the toxicity of cocaine effectively by preoperative administration of barbiturates. Secondary rise of temperature with spread or reactivation of the lesion following bronchoscopy was not seen in any of our cases. Although such complications have been reported by other authors, modern treatment methods probably prevented the occurrence of such unfortunate after-effects in our patients.

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Triboro Hospital, Jamaica, N. Y.

In Memoriam

HARRIS PEYTON MOSHER, M.D.,

1867-1954.

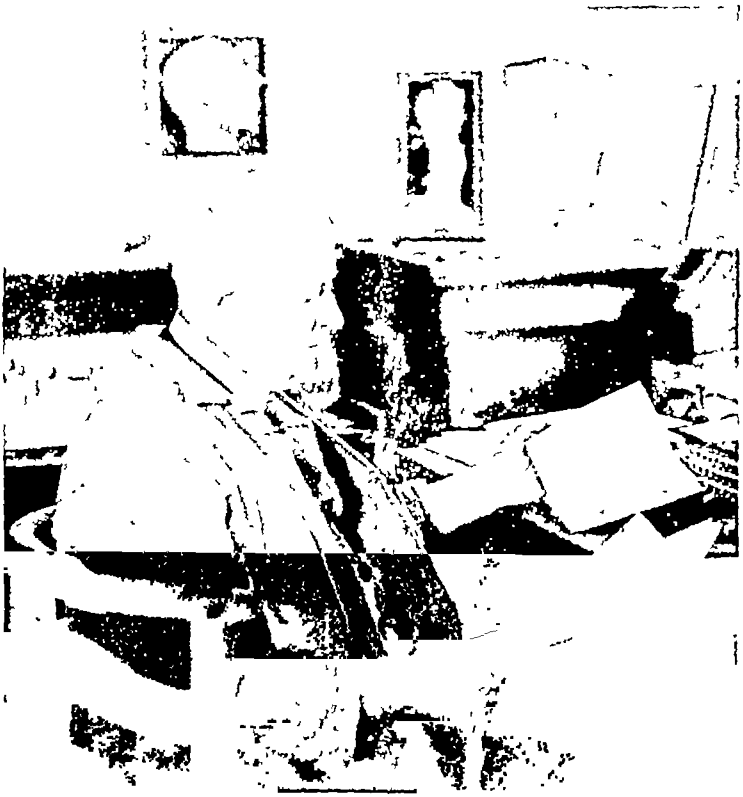
In the death of Dr. Harris P. Mosher in Salem, Massachusetts Hospital, Thursday, November 4, 1954, American otolaryngology lost another of its outstanding pioneers.

Dr. Mosher had lived over forty years in Boston and Marblehead, Mass.

Born in Woodford's, Maine, in 1867, he graduated from Boston Latin School, from Harvard College in 1892 and from Harvard Medical School in 1896, in the first class requiring four years for a degree. He became Harvard's first professor of otology and laryngology, the first full time teaching role in these specialties in this country, and continued in service there for over four decades.

It is given to few men to attain the leadership in their chosen field such as Dr. Harris P. Mosher achieved in his. By universal acclaim he was known as "The Chief." Some used this form of address as a friendly salutation, others in awe or respect; but everyone included in its use a feeling of reverence. His death brings to an end an era in otolaryngology the like of which will not be seen again.

No memorial can detail all of Dr. Mosher's achievements so no attempt will be made to do so here. Outstanding among them, however, is the fact that not only was he a member of all of the national otolaryngologic societies but he, in turn, served as president of each of them. When the American Board of Otolaryngology was formed he was chosen as its



president and served in that capacity for 25 years. While others worked hard for its position of excellence in the field of medicine, it was his driving zeal and high ideals which were responsible for the major share of its accomplishments. He was the recipient of many honors, among them several honorary degrees from leading American Universities and the Semon Medal from the Royal Society of Medicine of London, England. In 1931 Dr. Mosher was awarded the Gold Medal of the American Laryngological Association, and in 1939 a similar medal was awarded to him by the American Academy of Ophthalmology and Otolaryngology.

He will be remembered best, however, as a teacher of his specialty, at Harvard University, his Alma Mater, the Massachusetts General Hospital and the Massachusetts Eye and Ear Infirmary. He added fame to these illustrious institutions as citadels of learning. Testimony to his exceptional skill as a teacher is the number of his "boys" who have become chairmen of their departments in many schools and hospitals. He inspired more men to such attainments than any other teacher of his time, and scores of men who did not seek such positions owe their otolaryngologic successes to him.

He was an "inventor" as well as a doctor. He designed several instruments which added much to the surgical armamentarium of his day. In addition, he outlined several surgical procedures which remain as standards to the present. Among the most notable, being his technique in the ethmoid labyrinth and his method for the removal of safety pins swallowed by babies, for which he was given a Citation by the American College of Surgeons, in 1934. He was not a surgeon, with emphasis on fine detail as would suit the present era of fenestration surgery, but rather he was bold and brave, eminently fitted to attack the ravages of infection.

In 1905 Dr. Mosher became a collaborator on the staff of

THE LARYNGOSCOPE and his guiding counsel throughout these many years has been instrumental in attaining the enviable position this journal now holds in otolaryngologic literature.

Harris P. Mosher was an artist. This helped him immensely in his teaching endeavors, finding expression in telling illustrations. Many pictures of merit came from his pen or brush. He was an artist with a camera, and color photography furnished for him one of his great pleasures.

No memorial to Dr. Mosher would be complete without reference to his ability to find an appropriate word for a particular occasion. He was a past-master at repartee. The use of this gift enlivened many a national meeting when he rose to discuss a paper. His thinking was incisive, so he was quick to discuss a presentation if he thought it was lacking in merit; yet he was equally willing to give praise if it was deserved. At the last national meeting he attended, he went out of his way to praise a young man who had made his first national presentation. He lent rich color to all our gatherings.

There were many who thought Dr. Mosher was a rough, gruff man. This was because they did not know him, for he used an apparent austerity to cover one of the kindest and softest hearts we shall ever know. Many recipients of his graciousness and kindness testify to this. The number of young men to whom Dr. Mosher gave a friendly helping hand is legion.

Above all, he was loyal. Certainly he was loyal to the institutions he served. He was more than loyal to his "boys." His devotion to his wife, Helen, was magnificent. Finally, he was loyal beyond words to his chosen work; he gave his life for it, and its present enviable status is in no small measure due to his efforts.

A veteran of the Spanish-American War and World War I, he served overseas with the Harvard Hospital Unit in the former and in the Surgeon General's office in Washington, in the latter, holding the rank of Colonel.

Besides his many activities in the field of otolaryngology, Dr. Mosher found time to enjoy his memberships in several clubs, among them St. Botolph's, University, Boston Yacht and the Harvard Clubs of Boston and New York.

McCrae, at the close of World War I, wrote these words for those who *gave their lives on one of its famous battlefields:*

> *"To you from falling hands we throw the torch.*
> *Be yours to hold it high."*

Today, these words could well be coming to us from Harris P. Mosher. In the light of all that he was and all that he represented, it is our bounden duty to hold high indeed all that he passed on to us!

G. D. H.

UNIVERSITY OF FLORIDA MID-WINTER SEMINAR.

The Ninth Annual University of Florida Mid-winter Seminar in Ophthalmology and Otolaryngology will be held at the Sans Souci Hotel in Miami Beach, the week of January 17, 1955. The lectures on Ophthalmology will be presented on January 17, 18, and 19, and those on Otolaryngology on January 20, 21, and 22. A mid-week feature will be the Mid-winter Convention of the Florida Society of Ophthalmology and Otolaryngology on Wednesday afternoon, January 19, to which all registrants are invited. The registrants and their wives may also attend the informal banquet at 8 p.m., on Wednesday. The Seminar schedule permits ample time for recreation.

The Seminar lecturers on Ophthalmology this year are: Dr. William F. Hughes, Jr., Chicago; Dr. Phillips Thygeson, San Jose; Dr. James Allen, New Orleans; Dr. Walter H. Fink, Minneapolis; and Dr. Milton L. Berliner, New York. Those lecturing on Otolaryngology are: Dr. Paul Holinger, Chicago; Dr. Lawrence R. Boies, Minneapolis; Dr. Edmund P. Fowler, Jr., New York; Dr. Arthur W. Proetz, St. Louis, and Dr. David DeWeese, Portland, Oregon.

ANNOUNCEMENT.

An introductory course in Reconstructive Surgery of the Septum and External Nasal Pyramid will be given, under the direction of Dr. Maurice Cottle, March 19 through 26, 1955, at the University of Oregon Medical School. This will be sponsored by the Department of Otolaryngology.

INTERNATIONAL ASSOCIATION OF LOGOPEDICS AND PHONIATRICS.

The Tenth International Congress for Speech and Voice Therapy will take place in Barcelona, Spain, September 3 - 7, 1956.

Those practicing in the field of speech and voice therapy and in allied professions are invited to attend this Congress.

The following official reports will be presented: *Stuttering* — Dr. Wendell Johnson (Iowa); Neurology and Speech and Language Disorders — Dr. Antonio Subirana, (Barcelona); Non-verbal Tests of Intelligence and Maturity — Dr. Edeltrud Baar (Vienna); Amusia — Dr. Augusta Jellinek (New York).

Those submitting papers should notify the Organizing Secretary not later than January, 1956. No paper can be accepted unless a summary of not more than 100 words has been received by the Organizing Secretary before May 15, 1956. Time limit for paper — ten minutes. Articles that have been previously published cannot be accepted for the Congress.

The official languages of the Congress are English, French, German, and Spanish.

For further details, apply to the Organizing Secretary: Dr. J. Perelló, Provenza 319, Barcelona (9), Spain.

THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC., MEETINGS.

MID-WINTER.

Eastern Section Meeting..............................Friday, January 7, 1955
Philadelphia, Pa., Hotel Warwick

Triological Council Meeting...................Saturday, January 8, 1955
New York City, The Waldorf-Astoria

Western Section Meeting...Saturday-Sunday, Jan. 15-16, 1955
Los Angeles, Cal., The Town House

Southern Section Meeting..................Saturday, January 22, 1955
Charlottesville, Va., Medical School Auditorium

Middle Section Meeting.........................Monday, January 24, 1955
Detroit, Mich., Sheraton-Cadillac Hotel

SPRING.

American Board of Otolaryngology
Richmond, Va., March 6-10, 1955

All Society Meetings will be held at the Hollywood Beach Hotel, Hollywood, Florida.

American Laryngological Association..............March 13, 14, 1955
Triological Society......(mornings only) March 15, 16, 17, 1955
American Broncho-Esophagological Association
(afternoons only) March 15, 16, 1955
American Otological Society, Inc.......................March 17, 18, 1955

The Hollywood Beach Hotel (American plan) is a delightful place to hold the Spring Meetings — you may wish to bring the family along. The Hotel offers its guests such features as ocean bathing, deep sea fishing, 18-hole golf course and country club, tennis and many other activities.

For hotel reservations please communicate with Mr. John W. Tyler, Manager, Hollywood Beach Hotel, Hollywood, Florida.

TWENTY-FOURTH ANNUAL MID-WINTER CLINICAL CONVENTION IN OPHTHALMOLOGY AND OTOLARYNGOLOGY, JANUARY 17 THROUGH JANUARY 28, 1955.

The Research Study Club of Los Angeles announces its Twenty-fourth Annual Mid-Winter Clinical Convention in Ophthalmology and Otolaryngology, January 17 through January 28, 1955. For the Ear-Nose-Throat, the main Guest Lecturers will include Dr. Theodore E. Walsh, Professor and Head of the Department of Otolaryngology, Washington University, St. Louis; and Dr. John J. Conley, Chief of the Head and Neck Service, St. Vincent's Hospital, New York City, and Clinical Professor of Otorhinolaryngology, New York University, and other appointments.

Dr. Walsh has long been known to us not only as a professor, but also as a lecturer in our national societies, with particular interest in otologic and rhinologic problems.

Dr. Conley has done a great deal of work in tumors and plastic surgery of the head and neck, as well as being a well trained man in the broader field of our specialty.

Also serving on our program with Didactic Lectures will be Dr. Kenneth C. Brandenburg of Long Beach, Dr. J. C. Almy Harding of San Diego, and Dr. Paul J. Moses of San Francisco.

Instruction Courses in Otolaryngology will be provided by Dr. E. R. V. Anderson, Dr. Norman Jesberg, Dr. Harold Owens, and Dr. Manuel R. Wexler, all of Los Angeles, and Dr. Leo Shahinian of San Francisco.

The main Guest Lecturers for the Eye program will be Dr. Edmund B. Spaeth and Dr. Irving H. Leopold, both from the University of Pennsylvania Graduate School of Ophthalmology.

The first week will be devoted to the Ear-Nose-Throat—from Monday to Saturday, January 17 through January 22. The Eye week will cover Monday to Friday, January 24 through January 28. Those of us who confine our work to only one of these specialties may complete either subject in one week.

On Tuesday evening, January 18, the Annual Banquet of the Los Angeles Society of Ophthalmology and Otolaryngology will honor the Guest Lecturers of that week. This will be held at 7:00 p.m. in the Ball Room of the Elks Club.

On Wednesday afternoon of each week motion pictures will be shown, Ear-Nose-Throat subjects the first week, and Eye subjects the second week. These afternoons will also afford time for relaxation, sight-seeing, golf, radio broadcasts, and the enjoyment of points of interest in and about Los Angeles, including the famous Huntington Library with its collection of rare books and art.

The American Laryngological, Rhinological and Otological Society, Western Section, will hold its Annual Meeting at The Town House immediately preceding this Convention, Saturday and Sunday, January 15 and 16. On Saturday afternoon the scientific session is open to members and all interested otolaryngologists. Saturday evening there will be a dinner meeting for members of the Triologic Society only. Sunday morning, the second scientific session is open to all interested. The president of the Society, Dr. Kenneth M. Day, after attending this Society's Annual Meeting, will be with us during a portion of the Mid-Winter Course of the Research Study Club.

Unless you have your own plans for a place to stay, it is advised that you write for reservations at an early date to Mr. H. M. Nickerson, Manager of the Elks Club, 607 South Parkview Street, Los Angeles 57. He will endeavor to arrange suitable quarters for you in a conveniently located hotel, and will confirm the reservation by return mail.

Each applicant must be a member, in good standing, of the American Medical Association *in Order to Become Eligible* for attendance at the Convention. The fee for the entire two weeks, or any part of it, is $100.00 and includes the cost of all luncheons. We are informed that these dues are an income-tax deductible item, as they represent an annual membership fee. Make your Check payable to "Mid-Winter Clinical Convention," and mail to Pierre Violé, M.D., Treasurer, 1930 Wilshire Boulevard, Los Angeles 57. If anything prevents your attendance, this fee will be returned to you.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. D. E. Staunton Wishart, 170 St. George St., Toronto 5, Ontario, Canada.
Vice-President: Dr. Wm. J. McNally, 1509 Sherbrooke St., West Montreal 25, Canada.
Secretary-Treasurer: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Illinois.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: *Hollywood Hotel, Hollywood, Fla., March 17 (afternoon), 18 (forenoon and afternoon), 19 (forenoon), 1955.*

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. Henry B. Orton, Newark, N. J.
First Vice-President: Dr. Jas. H. Maxwell, Ann Arbor, Mich.
Second-Vice-President: Dr. Clyde A. Heatley, Rochester, N. Y.
Secretary: Harry P. Schenck, Philadelphia, Pa.
Treasurer: Fred W. Dixon, Cleveland, Ohio.
Librarian-Historian: Dr. Bernard J. McMahon, St. Louis, Mo.
Meeting: Hollywood Hotel, Hollywood, Fla., March 13-14, 1955.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Kenneth M. Day, 121 University Pl., Pittsburgh, Pa.
President-Elect. Dr. Dean M. Lierle, Iowa City, Iowa.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Eastern Section Meeting, Friday, January 7, 1955, Philadelphia, Pa., Hotel Warwick.
Triological Council Meeting, Saturday, January 8, 1955, New York City, The *Waldorf-Astoria.*
Western Section Meeting, Saturday - Sunday, January 15 - 16, 1955, Los Angeles, Cal., The Town House.
Southern Section Meeting, Saturday, January 22, 1955, Charlottesville, Va., Medical School Auditorium.
Middle Section Meeting, Monday, January *24, 1955,* Detroit, Mich., Sheraton-Cadillac Hotel.
Meeting: Hollywood Hotel, Hollywood, Fla., March 15-16-17, 1955, morning only.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. F. W. Davison, Danville, Pa.
Vice-Chairman: Dr. Guy L. Boyden, Portland, Ore.
Secretary: Dr. Hugh A. Kuhn, Hammond, Ind.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Algernon B. Reese, 73 East 71st St., New York 21, N. Y.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., October 9-15, 1955.

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Hotel Marshall, Richmond, Va., March 6-10, 1955.
Palmer House, Chicago, Ill., October, 1955.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Clyde A. Heatley, 11 No. Goodman St., Rochester, N. Y.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Square, Philadelphia, Pa.
Meeting: Hollywood Beach Hotel, Hollywood, Fla., March 15-16, 1955, afternoon only.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. John Louzan.
Vice-Chairman: Dr. Willard B. Walters.
Secretary: Dr. Jack L. Levine.
Treasurer: Dr. Russell S. Page, Jr.
Meetings are held on the third Tuesday of October, November, March and May, 7:00 P.M.
Place: Army and Navy Club, Washington, D. C.

GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Meeting: General Oglethorpe Hotel, Savannah, Ga., March 11-12, 1955.

THE GREATER MIAMI EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. George McKenzie, Dupont Medical Bldg., Miami, Fla.
Vice-President: Dr. Maurice Edelman, 605 Lincoln Road, Miami Beach, Florida.
Secretary: Dr. James H. Mendel, Jr., 7241 Red Road, Miami, Fla.
Meeting: Quarterly, at Seven Seas Restaurant, February, May, October, and December.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

President: Dr. Norman Leshin, 55 East Washington St., Chicago, Ill.
Vice-President: Dr. Henry B. Perlman, 950 East 59th St., Chicago, Ill.
Secretary-Treasurer: Dr. Stanton A. Friedberg, 122 So. Michigan Ave., Chicago, Ill.
Meeting: First Monday of each Month, October through May.

THE LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. W. L. Hughes, Lamar Life Bldg., Jackson, Miss.
Vice-President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

OTOSCLEROSIS STUDY GROUP.

President: Dr. Gordon D. Hoople, 1100 East Genesee St., Syracuse, N. Y.
Secretary: Dr. Lawrence R. Boies, Med. Arts Bldg., Minneapolis 2, Minn.
Meeting: Palmer House, Chicago, Ill., Oct. 9, 1955.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. F. Lambert McGannon, 14595 Madison Ave., Lakewood 9, Ohio.
Secretary-Treasurer· Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2, N. Y.
Meeting: Palmer House, Chicago, Ill., October, 1955.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. J. M. Tato, Azcuenaga 235, Buenos Aires, Argentina.
Executive Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U. S. A.
Meeting: *Fifth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.*
President: Dr. J. H. Font, Medical Arts Bldg., San Juan, P. R.
Time and Place: 1956, Puerto Rico.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.
Meeting:

THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. S. Fitz-Hugh, Charlottesville, Va.
President-Elect: Dr. H. L. Mitchell, Lexington, Va.
Vice-President: Dr. Marion K. Humphries, Charlottesville, Va.
Secretary-Treasurer: Dr. L. B. Sheppard, 301 Medical Arts Bldg., Richmond, Va.

Spring Meeting: Natural Bridge Hotel, Natural Bridge, Va., May 7-8, 1955.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President:Harold Owens, M.D.
Secretary-Treasurer: Robert A. Norene, M.D.

Chairman of Section on Ophthalmology: Sol Rome, M.D.
Secretary of Section on Ophthalmology: Wendell C. Irvine, M.D.

Chairman of Section on Otolaryngology: Max E. Pohlman, M.D.
Secretary of Section on Otolaryngology: Herschel H. Burston, M.D.

Place: Los Angeles County Medical Association Building, 1925 Wilshire Boulevard, Los Angeles 57, Calif.
Time: 6:00 P.M., first Thursday of each month from September to June inclusive—Ophthalmology Section. 6:00 P.M., fourth Monday of each month from September to June inclusive—Otolaryngology Section.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Harry Nievert, 555 Park Ave., New York (21), N. Y.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York (16), N. Y.
Meeting:

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. Cecil Swann, Asheville, N. C.
Secretary and Treasurer: Dr. Geo. B. Ferguson, Durham, N. Car.
Meeting:

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. David S. Asbill, Columbia, S. Car.
Vice-President: Dr. John McLean, Greenville, S. Car.
Secretary-Treasurer: Dr. Roderick Macdonald, Rock Hill, S. Car.
Meeting:

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.
Secretary-Treasurer: Dr. John F. Tolan, 3419 47th Ave., Seattle (5), Wash.
Meeting:

THE RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Chairman: Dr. Isaac H. Jones, 635 S. Westlake, Los Angeles, Calif.
Treasurer: Dr. Pierre Violé, 1930 Wilshire Blvd., Los Angeles, Calif.
Program Chairmen:
  Otolaryngology: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.
  Ophthalmology: Dr. Harold F. Whalman, 727 W. 7th St., Los Angeles, Calif.

Mid-Winter Clinical Convention annually the last two weeks in January at Los Angeles, Calif.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Chas. C. Grace, 145 King St., St. Augustine, Fla.
President-Elect: Dr. Jos. W. Taylor, 706 Franklin St., Tampa, Fla.
Secretary-Treasurer: Dr. Carl S. McLemore, 1217 Kuhl Ave., Orlando, Fla.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President: Dr. William J. Hitschler.
Vice-President: Dr. Chevalier L. Jackson.
Treasurer: Dr. John J. O'Keefe.
Secretary: Dr. Joseph P. Atkins.
Historian: Dr. Herman B. Cohen.
Executive Committee: Dr. Thomas F. Furlong, Jr., Dr. William A. Lell, Dr. Harry P. Schenck, Dr. Benjamin H. Shuster, ex-officio.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Vice-Chairman: Dr. K. W. Cosgrove, 111 E. Capitol Ave., Little Rock, Ark.
Secretary: Dr. F. A. Holden, Medical Arts Bldg., Baltimore, Md.
Meeting:

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James K. Stewart, Wheeling, W. Va.
Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. C. Otrich, Belleville, Ill.
President-Elect: Dr. Phil R. McGrath, Peoria, Ill.
Secretary-Treasurer: Dr. Alfred G. Schultz, Jacksonville, Ill.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Oscar Marchman, Jr., Dallas, Texas.
Secretary-Treasurer: Dr. Morris F. Waldman, Dallas, Texas.

BALTIMORE NOSE AND THROAT SOCIETY

Chairman: Dr. Albert Steiner, 1308 Eutaw Pl., Baltimore, Md.
Secretary-Treasurer: Dr. Walter E. Loch, 1039 No. Calvert St., Baltimore, Maryland.

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDICO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Salvador Mixco Pinto.
Secretary: Dr. Daniel Alfredo Alfaro.
Treasurer: Dr. Antonio Pineda M.

CANADIAN OTOLARYNGOLOGICAL SOCIETY
SOCIETE CANADIENNE D'OTOLARYNGOLOGIE

President: Dr. G. Edward Tremble, 1390 Sherbrooke St., West, Montreal, P. Q.
Secretary: Dr. G. Arnold Henry, 170 St. George St., Toronto, Ontario.
Meeting: Bigwin Inn, Lake of Bays, Muskoka, Ontario. June 16 to June 18, 1955.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

President: Dr. Cesar LaBolde, Mexico, D. F.
Vice-President: Dr. M. Gonzalez Ulloa, Mexico, D. F.
Secretary: Dr. Juan de Dios Peza, Mexico, D. F.

FEDERACION ARGENTINA, DE SOCIEDADES DE OTORRINOLARINGOLOGIA.

Secretario del Exterior: Dr. Juan Manuel Tato.
Sub-Secretario del Exterior: Dr. Oreste E. Bergaglio.
Secretario del Interior: Dr. Eduardo Casterán.
Sub-Secretario del Interior: Dr.Atilio Viale del Carril.
Secretario Tesorero: Dr. Vicente Carri.
Sub-Secretario Tesorero: Dr. José D. Suberviola.

ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

Presidente: Dr. J. Abello.
Vice-Presdente: Dr. Luis Suñe Medan.
Secretario: *Dr. Jorge Perelló, 319 Provenza*, Barcelona.
Vice-Secretario: Dr. A. Pinart.
Vocal: Dr. J. M. Ferrando.

SOCIEDAD NACIONAL DE CIRUGIA OF CUBA.

Presidente: Dr. Reinaldo de Villiers.
Vicepresidente: Dr. César Cabrera Calderin.
Secretario: Dr. José Xirau.
Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Andre Soulas, Paris, France.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa. U. S. A.
Meeting:
Time and Place:

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER — CAMPINAS.

President: Dr. Heitor Nascimento.
First Secretary: Dr. Roberto Barbosa.
Second Secretary: Dr. Roberto Franco do Amaral.
Librarian-Treasurer: Dr. Leoncio de Souza Queiroz.
Editors for the Archives of the Society: Dr. Guedes de Melo Filho, Dr. Penido Burnier and Dr. Gabriel Porto.

SOCIEDAD CUBANA DE OTO-LARINGOLOGIA.

President: Dr. Reinaldo de Villiers.
Vice-President: Dr. Jorge de Cárdenas.
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